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THE VENOM YIELDS OF COMMON AUSTRALIAN POISONOUS SNAKES IN CAPTIVITY.

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AND

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THE present communication should be regarded as an addendum to the classical paper on this subject by Hamilton Fairley and Splatt.[1] They observed the primary yields and, during a period of about four months, the secondary yields at monthly and in some cases fortnightly intervals of limited numbers of marked individual tiger snakes, death adders, copperheads and black snakes. They were able to correlate the size of the yields with the length of the snakes and the distance separating the fangs.

We have been concerned primarily with the collection of venom and, except in the case of very large snakes, have not recorded the size of the snakes or collected individual yields separately. From October, 1928, to March, 1934, we have milked at approximately monthly intervals a population of snakes housed at the Melbourne Zoological Gardens varying during the course of each year from less than 100 to over 300 in number. We have carried out a good many thousand milkings upon 2,000 venomous snakes, many of which have been in captivity a year or more. We can supply from our data supplementary information concerning the average venom yields in captivity of large numbers of snakes of each of the species dealt with by Fairley and Splatt, the mortality of snakes in captivity and the rate at which the average venom yield may fall off under these conditions. Our figures for the highest individual yield of each species naturally exceed those of Fairley and Splatt. Finally, we can pro-

vide some information about the venom yield of the common brown snake and of the melanotic island form of the tiger snake.

The Venom Yield of the Tiger Snake (Notechis Scutatus).

During the period October, 1928, to March, 1934, we have carried out over 3,500 milkings on approximately 870 tiger snakes. The average yield for 3,214 milkings of which we have accurate record is 0·0276 gramme of dry venom.

Fairley and Splatt obtained a higher average venom yield (0·036 gramme) in 62 observations on 20 healthy tiger snakes of average length 997 millimetres (3 feet 3¼ inches) and a much lower yield (0·010 gramme) in 134 observations on 60 snakes (of average length 896 millimetres) which were in poor condition and heavily infested with lung fluke. Our figures include yields from small snakes and snakes in poor condition, many of these latter "nil" yields. When we have had large numbers of snakes in captivity it has not always been possible to provide sufficient food for them. Fairley and Splatt's average figure for healthy snakes probably approaches much more nearly to that which would be obtained if snakes of this species were adequately fed and kept under more natural conditions.

The highest average yield for any large group of tiger snakes on any one occasion was 0·054 gramme for 58 snakes in February, 1933, and three weeks later the same snakes yielded an average of 0·050 gramme at their second milking. In Table I are set out a number of the average yields we have obtained for large groups of tiger snakes. In these large unselected groups it may be assumed that the average size is a fair average for the species.

TABLE I.

Venom Yields by Large Groups of Tiger Snakes.

Month and Year.	Number of Snakes.	Total Venom Yield in Grammes.	Average Yield in Grammes.
January, 1929	60	1·871	0·031
November, 1929	122	3·867	0·032
February, 1930	79	3·062	0·039
February, 1931	64	2·536	0·039
January, 1932	58	2·034	0·035
December, 1932	113	3·349	0·030
February, 1933	58	3·163	0·054
January, 1934	106	3·698	0·035

The highest individual yield recorded by Fairley and Splatt was 0·155 gramme in a snake 1,190 millimetres (nearly 3 feet 11 inches) long. The highest single yield we have observed was 0·189 gramme in a tiger snake 1,582 millimetres (5 feet 2¼ inches) long. Incidentally the longest tiger snake we have had in our possession measured 1,663 millimetres (5 feet 5½ inches).

The longest observed series of milkings on a single snake extended over a year, and the yields obtained in eight milkings during this period were 0·150, 0·089, 0·103, 0·091, 0·081, 0·075, 0·063 and 0·043, giving an average of 0·087 gramme of dry venom.

Snakes of this species have not survived any great length of time in captivity. This is illustrated by the fall in numbers of snakes in captivity which regularly occurs during the late autumn and winter months. In 1932, on April 26, we milked 132 tiger snakes; on May 24, 90; on June 20, 75; and at the end of August, only 36. The mortality during four months was over 72%. The average venom yields during this period were fairly steady: 0·025, 0·026, 0·033 and 0·028 gramme of dry venom. This year, in a population kept up to over a hundred by the addition of freshly caught snakes, our losses in March amounted to 40 and in April to over 30 deaths.

The causes of this mortality are overcrowding, inanition, natural diseases (for example, infestation with lung fluke (*Dolichopera maculpini*) and "tail disease") and diseases peculiar to captivity, such as "cancre". A number of deaths occur in the early months of the year in females who have recently given birth to young.

Under the conditions obtaining at the Melbourne Zoological Gardens, where all the snakes are kept in concrete floored cages, which are neither drained nor readily disinfected and where no open air snake pit is available, these snakes rarely survive longer than a year in captivity. We had one series of healthy snakes, twenty-six in number, caught at the end of a season, most of which survived for eighteen months. On the other hand, some snakes kept singly, after excision of their venom glands, in petrol tins with wire gauze lids lived for two years.

It has recently been shown by workers at this Institute[2] that whereas freshly caught snakes have few organisms in their mouths, in captivity they rapidly acquire a very large oral bacterial population.

To achieve better average venom yields and to diminish mortality in captivity an adequately drained and extensive open air snake park is essential, so as to avoid overcrowding and to make the conditions of captivity as nearly natural as possible. This has not been possible for economic reasons. Our attempts to disinfect the cages have not been adequate, except in the middle of winter, because throughout the summer the space available has always been overcrowded. The cages are cleaned out mechanically every month when the snakes are milked, but it is not then possible to disinfect them, since they are vacant for only half an hour or so.

Overcrowding diminishes the average yield of this species, particularly if there are several big snakes in a cage, because some of them are certain to be aggressive and spend much time snapping and biting at their fellows and at inanimate objects.

The Venom Yields of the Black Tiger Snake (Notechis Scutatus var. Niger).

We obtained in February, 1931, forty-nine specimens of the black tiger snake, a melanotic form of *Notechis scutatus*, from Chappell Island, Bass Strait. The venom yields for the first six months of captivity have already been described (Kellaway and Thomson[3]). The further history of these snakes is of some interest, since the last survivor did not die till February of this year. The female snakes gave birth to young in March, 1931, but have

not done so in succeeding years. The original population of 49 was reduced by August 28, 1931, to 33. In January of the following year there were still 30 alive. By December, 1932, there were eight survivors, though four had been sent to the London Zoological Gardens and some had been used for experiments. In February, 1933, there were still eight survivors, but all except one died during the winter of 1933, the last dying shortly after being milked early in February, 1934. The venom yields from August, 1931, onwards are set out in Table II, which is a continuation of Table I of Kellaway and Thomson's paper.

TABLE II.

Venom Yields of Black Tiger Snakes in Captivity.

Date.	Number of Snakes.	Total Yield of Venom.	Average Yield in Grammes.
October 2, 1931 ..	33	2·189	0·066
November 6, 1931 ..	31	2·277	0·073
December 15, 1931 ..	31	2·779	0·090
January 2, 1932 ..	30	2·702	0·090
April 26, 1932	27	1·814	0·067
May 24, 1932	23	1·044	0·045
June 24, 1932	23	1·031	0·045
September 6, 1932 ..	13	0·773	0·059
December 15, 1932 ..	8	0·423	0·053
February 8, 1933 ..	8	0·484	0·060

The fall in numbers does not truly represent the mortality in captivity, because a few of these snakes were used for experiment, killed for morphological studies or sent to other zoological gardens.

In Table II only those "milkings" have been omitted in which the number of snakes dealt with was uncertain, and a few of the last series in which only one snake was "milked". The average of these yields, together with those earlier recorded, a total of 516 milkings, is 0·07 gramme. The average length of these snakes was 1,375 millimetres (4 feet 6¼ inches) and the largest 1,780 millimetres (5 feet 10 inches). They were extremely well nourished when captured and their mean weight was 1·06 kilograms. A good many of them were pregnant females. They were very healthy, except for the presence of tail disease, an infection of uncertain origin, which often results in snakes losing the terminal part of the tail. Six of these snakes had well healed truncated tails, and in several others active disease was present.

The largest individual venom yield observed was 0·3886 gramme.

These snakes did surprisingly well in captivity, despite the difficulty of providing an adequate food supply. In their island home they had doubtless had in the season an unrestricted supply of young mutton birds, which accounted for their excellently nourished condition when captured. We fed them for the most part on small rats.

The Venom Yields in the Death Adder (Acanthophis Antarcticus).

Fairley and Splatt obtained the individual primary yields and three further secondary yields at monthly intervals of 48 death adders. These they divided into a group of 41 snakes between 45·5 and 73·0 centimetres long and a group of seven snakes between 32·5 and 45·5 centimetres in length. The average venom yield in the first group was 0·0649 gramme and in the second 0·0097 gramme of dry venom.

The average figures recorded here are derived from 926 milkings of 360 death adders. Some groups of milkings have been omitted because of uncertainty about the number of snakes milked. The average venom yield for our series was 0·040 gramme of dry venom. This figure is much lower than that of Fairley and Splatt's series, partly because we have included a number of very small yields obtained after some months in captivity, and partly because we have not been able to procure sufficient food when, as at one period, we had over 200 of these snakes in captivity at one time.

Fairley and Splatt's series of adult snakes showed only a slight falling off in the venom yields after the primary milking; ours, on the contrary, exhibits very striking decrease, as is illustrated by the following series, which incidentally gives some idea of the death rate in captivity. On March 7, 1929, 95 adders at a primary milking yielded an average of 0·057 gramme; on April 2, 87 snakes gave 0·026 gramme; on April 24, 74 snakes gave 0·022 gramme; on May 17, 74 snakes gave 0·024 gramme; on June 30, 67 snakes gave an average of 0·033 gramme; and on July 30, 28 snakes gave an average of 0·049 gramme of dry venom. The closer interval between the earlier milkings accounts in part for the rapid falling off of the venom yield.

In no case were the average primary yields of our unselected groups of death adders so high as the average primary yield of Fairley and Splatt's two groups combined, namely, 0·0685.

The average primary yield of 91 adders on February 20, 1930, was 0·062 gramme, and the average primary yield of 134 adders on April 15 of the same year was 0·060 gramme. The largest individual yield observed by Fairley and Splatt was 0·2356, and our largest was 0·252 gramme.

The Venom Yields of the Copperhead (Denisonia Superba).

Since October, 1928, we have had approximately 590 copperheads in captivity. In 1,940 milkings of which we have accurate records, the average yield was 0·0215 gramme of dry venom, somewhat more than Fairley and Splatt's average 0·0185 gramme for 26 copperheads of average length 1,000 millimetres, which did not include any really large snakes.

To give an idea of the size to which this species grows, we may instance a single catch from Mount Gambier in February, 1931. This consisted of 26 snakes, the largest of which was 1,438 millimetres (4 feet 8½ inches) long and weighed 1·085 kilograms. Other large snakes in this catch were 1,320, 1,317, 1,308, 1,305 millimetres long, weighing 1·025, 1·075, 0·935 and 0·895 kilograms. The largest individual primary venom yield in this group was 0·227

gramme and the next 0·218 gramme. The average primary venom yield of 13 of these snakes on February 13 was 0·092 gramme. On March 5, at their second milking, the average of 23 remaining snakes (average length 1,156 millimetres (3 feet 9½ inches) was 0·040 gramme.

In view of the large venom yields of these freshly caught snakes, it may appear surprising that the average venom yield over the whole period is so small. This is attributable to the fact that this species does not do well in captivity, the venom yields rapidly declining almost to nothing.

The copperhead is an extremely muscular snake and bites very strongly on the glass vessels used to collect the venom, damaging thereby its mandibular teeth, which are also further injured during the process of making the snake let go. This species does better if "milked" at less frequent intervals—six weeks instead of the usual three or four.

The mortality rate and decline of yield in captivity are illustrated by the following: On November 26, 1928, the average primary yield of 56 Mount Gambier copperheads was 0·040 gramme; on December 17, 49 gave an average yield of 0·024; and on January 9, 1929, 40 gave an average yield of 0·030 gramme. On January 29, 38 gave an average yield of 0·019 and 22 freshly caught specimens of smaller size gave an average yield of 0·032. On March 8, 46 survivors yielded an average of 0·017 gramme; on March 26, 38 survivors yielded an average of 0·013 gramme. On April 24, 25 copperheads yielded 0·019 gramme; on May 17, 20 yielded 0·011 gramme; and on May 30 these 20 survivors yielded an average of 0·018 gramme of dry venom.

The average yields recorded by Fairley and Splatt and by ourselves do not give a satisfactory picture of the venom-producing capacity of this snake under natural conditions. Generally speaking, the average primary yield of freshly caught snakes gives the best indication, but even this is too low a figure, since much venom is lost by the reptiles in biting one another in the bags after capture.

It is fortunate that persons are rarely bitten by snakes of this species, since the venom is extremely potent. Copperheads are rather slow compared with the other common snakes and are rarely aggressive unless cornered.

The Venom Yields of the Black Snake (Pseudechis Porphyriacus).

During the period under review we have had in captivity approximately 170 black snakes. The average venom yield in 579 milkings of which we have exact records of the numbers of snakes milked was 0·030 gramme, which is somewhat less than Fairley and Splatt's average for six snakes kept five months in captivity of 0·035 gramme. The snakes usually gave most at the primary milking and the values fell off somewhat at subsequent milkings. They seldom survived longer than seven or eight months in captivity. The following figures illustrate the mortality in captivity and the fall in average venom yield. A group of 13 snakes on January 22, 1932, gave an average of 0·059 gramme; on March 7, 12 snakes gave 0·041 gramme; on April 26, 10 snakes gave an average of 0·030; on May 5, 5 snakes yielded an average of 0·024; on June 20, the three survivors yielded an average of 0·026 gramme; and on July 15, these three snakes gave an average of 0·023 gramme of dry venom.

Between November, 1933, and the end of January, 1934, we obtained by purchase 76 black snakes. By the end of February these were reduced to 63. On March 23, 1934, there were 51; on April 14, 47; on May 3, 31; and on May 31, only 18 remained alive. A number of these snakes were females who had given birth to young in captivity and shortly afterwards died. The average yields obtained in January and the succeeding months were 0·041, 0·032, 0·045, 0·028, 0·021 and 0·028.

We have not kept any records of individual yields of snakes of this species. The highest average yields we have observed were of two specimens from Queensland which gave an average primary yield of 0·143 and at a milking two months later an average of 0·148 gramme. With this exception the average primary yield of 0·059 for 13 snakes, mentioned above, is the highest we have observed.

The Venom Yield of the Brown Snake (Demansia Textilis).

During the period under review we have had about 50 brown snakes in captivity. One snake, nearly seven feet in length, yielded 0·0672 gramme at a primary milking (0·045 at the first bite) on November 6, 1931, and on January 22, 1932, yielded 0·038 gramme. Excluding the yields from this large snake, the average yield in 126 recorded milkings, including those already placed on record by Kellaway,[4][5] was 0·002 gramme. Apart from the venom yields of our single very large brown snake, yields of 0·0099, 0·0074, 0·0072 and 0·0072 gramme have been the largest obtained from individual snakes.

We have summarized in Table III the data we have presented here for comparison with Table XXIV in Fairley and Splatt's paper.

TABLE III.

The Venom Yields in Different Species of Australian Snakes.

Species of Snake.	Number Examined.	Number of Milkings Recorded.	Average Yield in Captivity in Grammes.	Highest Average Yields.		Highest Individual Yield.
				Number of Snakes.	Average Yield.	
Brown snake	50	126	0·002			0·0672[1]
Black snake	170	579	0·030	13	0·059	0·148[2]
				54	0·041	
Copperhead	590	1,940	0·0215	13	0·092	0·227
				56	0·040	
Black tiger snake ..	49	516	0·0705			0·3886
Death adder	360	926	0·040	95	0·057	0·252
				91	0·062	
				134	0·060	
Tiger snake	870	3,214	0·0276	64	0·039	0·189
				29	0·039	

[1] Not included in the average.

[2] Average of two Queensland specimens.

These figures for the average yield at a milking of Australian snakes in captivity and those of Fairley and Splatt cannot be regarded as being equivalent to the average yield at a single bite of these species in a natural state. Actually the yield at a first bite by fresh snakes with fully charged venom glands is probably somewhat greater than the average venom yield in captivity. This is suggested by Fairley and Splatt's twelve observations on death adders (average 0·046 gramme) and six on tiger snakes (average 0·0386 gramme). No figures for the yields in a large series of single bites by freshly caught snakes are available.

The figures given here for average yields on milking are of use only as indicating the total yields of venom that are likely to be obtained for the preparation of antivenene or other purposes from a collection of average sized snakes. For this purpose perhaps these supplementary figures are more useful than those in Fairley and Splatt's paper, since they are obtained from a very large population kept in captivity for as long as possible under the conditions obtaining.

The high mortality observed and the decline in the average venom yields in the later months of captivity suggest that these conditions are far from optimal and that they must be improved if the average yield is to be increased. It is not worth while to handle large numbers of small snakes, and far better results with less trouble and risk could be obtained by keeping only the larger specimens for the collection of venom. Loss by indiscriminate biting when large snakes are herded together could probably best be avoided by keeping the snakes in separate compartments, though this is not practicable if large numbers have to be handled. The high incidence of mouth infection under the present conditions could probably be overcome only by keeping the reptiles in a properly drained open air park for a large part of the year.

Acknowledgements.

Our thanks are due to the President, Council and Director of the Melbourne Zoological Gardens, where our snakes have been housed, and to Mr. Tom Eades, who has handled the snakes for us during the process of "milking".

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RELATIONS BETWEEN THE SIZE AND STATE OF NUTRITION OF MOTHERS, THE PROPERTIES OF THEIR MILK, AND THE WEIGHT OF THEIR INFANTS.

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THE size which an individual attains at adult development is largely determined by the size of his parents. Pearson and Lee (1903), for example, showed long ago the existence of a positive correlation between the statures of parents and their adult offspring.

The existence of a positive correlation between the size of parents and their adult offspring does not necessarily imply the existence of such a correlation at every stage of the development of the offspring. Murray (1924) has examined the correlation between the heights of *primiparæ* and the lengths and weights of their infants at birth. Although she showed that there are statistically significant positive correlations between these quantities, the correlation coefficients are so low that their interpretation is difficult.

The distinct correlation between the size of parent and offspring which is reached at maturity must, however, show itself at some stage of growth earlier than this. It was thought that by dividing a number of mothers into sufficiently widely separated groups with regard to their stature, some relation between their stature and the weights of their infants during the first six months of life might become apparent. For the purpose of the present examination, therefore, the mothers have been divided into three obvious groups: (i) women obviously below the average stature; (ii) women not differing obviously from the average—the majority of the subjects; (iii) women obviously above the average stature.

The 145 women subdivided in this way were all in normal health; all were completely suckling normal infants; otherwise they were unselected. A relation has already been shown to exist between the properties of the milk of these women and the weights of their infants (Wardlaw and Dart, 1932).

Although a relation has also been shown to exist between the weights of infants and the numbers of children in the families (Wardlaw and Dart, 1932), women with different numbers of children have not been treated separately. Their distribution in the different stature groups is sufficiently similar to allow of this factor being neglected in a preliminary survey of such a small number of individuals. The average numbers of children for the three groups are 2·0, 2·5 and 2·0 respectively.

To enable comparisons to be made between the weights of infants of different sexes and ages, their

weights have been expressed as percentages of certain standards for the same sex and age. These percentages are called the relative weights. The standard values used are those given by Brailsford Robertson (1915) for South Australian infants. These figures agree very closely with comparable observations on New South Wales infants (Wardlaw, 1926).

If any correlation exists between the stature of mothers and the relative weights of their infants, a correlation is to be expected also between the stature of the women and the properties of their milk, since a correlation has been found between the properties of the milk and the relative weights of the infants. The data have therefore been examined from this point of view also.

The position with regard to the state of nutrition of mothers and the weights of their infants is quite different. Several observers have reported that under-nourished women give birth to infants of normal weight and length (Momm, 1916; Rabnow, 1916; Rüge, 1916; Schmidt, 1918; Soergel, 1918). The literature dealing with this subject in connexion with the effect of prolonged under-nutrition on the peoples of Central Europe during the Great War has been well summarized by Murray (1924). Her investigations confirm the independence of the weight and length of an infant at birth of the state of nutrition of the mother. It appears also that under-nourished mothers may for a limited period secrete milk adequate to the needs of their infants, even if the maternal tissue must be drawn upon to supply the constituents needed (Mössmer, 1916; Kütting, 1925). Similar observations have been made on lactating cows. The possibility of "milking the flesh off the bones" is well known to dairymen.

The women examined in the present investigation were chosen as normal with regard to their state of nutrition as well as in other respects. None of them was definitely under-nourished, nor did any show a degree of obesity which could be regarded as pathological. They showed, however, a considerable range in their state of nutrition. As for stature, each could readily be placed in one of three large groups: (i) those obviously thinner than the average; (ii) the majority of the women who showed no striking departure from the average; (iii) those obviously fatter than the average.

The data have been examined for the existence of a correlation between the state of nutrition of the women and the relative weights of their infants, and between the state of nutrition and the properties of their milk. For these groups also the distribution of the women with different numbers of children among the three groups does not vary enough to require this factor to be taken into account. The average numbers of children for the three groups are: 2·7, 2·1 and 2·0 respectively.

Stature and Weight of Infant.

The serial numbers of the women in the three groups are given in Table I. From these the details with regard to each may be obtained by reference to the papers of Wardlaw and Dart (1926, 1932).

TABLE I.

Serial Numbers of Women in Different Stature Groups.

Short:	2, 6, 7, 8, 22, 23, 44, 46, 50, 54, 55, 57, 62, 63, 76, 80, 82, 83, 87, 92, 99, 101, 108, 113, 115, 120, 123, 124, 140, 144, 154.
Average:	1, 4, 11, 12, 13, 16, 18, 20, 26, 27, 29, 30, 31, 32, 33, 34, 36, 40, 41, 43, 47, 48, 49, 51, 52, 53, 59, 60, 61, 64, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 77, 78, 79, 81, 85, 86, 88, 89, 90, 93, 94, 95, 96, 97, 103, 105, 106, 107, 109, 110, 112, 116, 117, 122, 125, 127, 128, 129, 130, 131, 133, 134, 137, 141, 143, 145, 146, 147, 148, 149, 150, 151, 152, 153, 155, 156, 158, 159, 160, 161, 163.
Tall:	3, 5, 9, 10, 15, 24, 25, 28, 37, 39, 56, 98, 100, 102, 104, 114, 118, 142, 157, 162, 164.

In Table II the mean relative weights of the infants and the mean values of the properties of the milk of the women in the three groups are given.

TABLE II.

Mean Values of Relative Weights of Infants, and of Properties of Milk of Short, Average and Tall Women.

Observation.	Short.	Average.	Tall.
Number	32	91	22
Relative weight of infant	93·2	100·3	100·6
Volume of milk sample in cubic centimetres	53·9	65·3	68·9
Weight of fat in grammes	2·59	3·05	3·12
Percentage of fat	4·81	4·67	4·54
Weight of solids not fat	4·77	5·79	6·11
Percentage of solids not fat	8·85	8·87	8·87

The figures in Table II show distinct difference between the mean values for the different groups. As the number of results is small, conclusions cannot be drawn from these differences without first examining their validity. This has been done by observing the distribution of the individual results about certain arbitrary values. The values chosen are the mean values for the "average" group of women. The ratio of the differences to their standard deviations give a measure of the odds against the occurrence of a difference in the direction observed being due to the chances of sampling (see Dunn, 1929). The numbers of results above the arbitrary value and the odds to one against the difference from the average group being due to chance are shown in Table III. The total number of results in each group is shown in parentheses.

TABLE III.

Stature of Mother, Weight of Infant, and Properties of Milk: Distribution of Results and Significance of Differences.

Observation.	Short (32).		Average (91).		Tall (22).
	Number.	Odds.	Number.	Odds.	Number
Relative weight	10	165·0	50	1·8	13
Volume of milk	11	6·1	41	1·7	9
Fat (weight)	9	13·3	38	2·3	11
Fat (percentage)	20	7·3	46	0·0	11
Solids not fat (weight)	11	4·5	39	3·2	8
Solids not fat (percentage)	15	4·9	52	1·3	12

The data in Table III confirm on the whole the impression given by the mean figures in Table II. It is evident that the small differences between the average and the tall groups are without significance.

The differences between the short and the average women are greater, and so are their significances. The odds are 165 to 1 in favour of there being a real tendency for the infants of the short women to be lighter than the infants of the average women during the first six months of life.

Stature and Properties of Milk.

The quantities of milk and of its constituents secreted in a given period must be considered separately from the concentrations at which the constituents occur. The quantities given are those secreted in a period of four hours.

The mean figures in Table II show only small differences in the yield and composition of the milk of the tall and average women. The differences between the means of the short and the average women are greater. The mean volume of milk and the mean weights of its constituents are smaller for the short women. The figures in Table III show that little significance can be attached even to the differences between these groups. The differences are in the direction which would be expected from the correlation between weight of infant and yield of milk.

If the volume of milk and the weights of its constituents may be regarded as independent variables, the significance of the simultaneous occurrence of all the differences in the same direction is much more than that of a single difference. The significance of the differences is further increased when the short women are compared with the average and tall women considered as one group. There appears to be a slight tendency for undersized women to yield less milk in a given time than larger women.

The differences between the mean concentrations of fat and of solids not fat of the short and the average and tall women are small, but it is interesting to notice that the difference for the concentration of fat is in the opposite direction to that for the quantity. The significance of this difference is negligible, but the figures are in harmony with the well known fact that the milk of small animals tends to be richer in fat than the milk of large animals (Raudnitz, 1909).

State of Nutrition and Weight of Infant.

The serial numbers of the subjects, divided into the three groups mentioned with respect to their state of nutrition, are given in Table IV.

TABLE IV.

Serial Numbers of Women in Different Nutrition Groups.

Thin:	6, 8, 9, 20, 22, 24, 34, 40, 44, 46, 50, 51, 54, 56, 59, 60, 61, 72, 76, 78, 80, 93, 100, 101, 104, 107, 108, 112, 113, 114, 117, 123, 124, 127, 130, 137, 152.
Average:	1, 2, 3, 5, 7, 10, 11, 12, 13, 14, 15, 16, 18, 23, 25, 26, 27, 28, 30, 31, 32, 33, 36, 37, 41, 43, 47, 48, 49, 52, 53, 55, 57, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 73, 74, 75, 81, 82, 83, 85, 86, 87, 88, 89, 90, 92, 94, 95, 96, 97, 98, 99, 102, 103, 105, 109, 110, 115, 116, 118, 120, 122, 125, 128, 129, 131, 133, 134, 136.
Fat:	140, 142, 143, 147, 148, 149, 151, 153, 154, 155, 157, 158, 159, 160, 161, 162, 163, 164.
Fat:	4, 29, 39, 79, 106, 141, 144, 145, 146, 150, 156.

The mean relative weights of the infants of the women in the three groups are shown in Table V.

TABLE V.

Average Values of Relative Weights of Infants, and of Properties of Milk of Women of Different States of Nutrition.

Observation.	Thin.	Average.	Fat.
Number	37	97	11
Relative weight of infant	95·6	100·1	102·2
Volume of sample	59·9	65·2	56·2
Weight of fat	2·69	3·10	2·43
Percentage of fat	4·49	4·75	4·32
Weight of solids not fat	5·13	5·77	4·73
Percentage of solids not fat	8·57	8·84	8·42

The mean relative weight of the infants of women in the thin groups is distinctly lower than that of the infants of women in the other two groups. The distribution of the individual figures about the mean for the average groups and the significance of the differences are shown in Table VI in the same way as in Table III.

TABLE VI.

State of Nutrition of Mother, Weight of Infant, and Properties of Milk: Distribution of Results and Significance of Differences.

Observation.	Thin (37).		Average (97).		Fat (11).
	Number.	Odds.	Number.	Odds.	Number.
Relative weight	19	1·2	48	1·0	6
Volume of milk	13	5·2	44	0·0	5
Fat (weight)	13	2·3	41	1·2	4
Fat (percentage)	13	32·3	51	6·1	4
Solids not fat (weight)	19	4·9	42	2·0	4
Solids not fat percentage	12	>1000·0	60	1·2	7

The distributions in this table do not confirm the impression given by the mean values. The differences between the different groups are seen to be quite without significance and are due to the uneven distribution of a small proportion of exceptional values.

Within a normal range the state of nutrition of the mother appears to have no relation to the weight of her infant.

State of Nutrition and Properties of Milk.

The mean values of the volume of milk secreted in four hours and of the quantities and concentrations of its principal constituents are shown in Table V. The mean values for the average group are greater than for the other two groups. The distributions given in Table VI show, however, that no significance can be attached to these differences, except for the percentage of solids not fat. This is definitely lower for the thin women than for the women in the other two groups. There is perhaps a tendency for the percentage of fat in the milk of the thin women to be lower also. The thin women therefore tend to secrete a less concentrated milk than the better nourished women, but, as with the weights of their infants, there is no evidence that the weight of the milk constituents secreted in a given time is any less than that in better nourished women.

Summary.

The breast-fed infants of undersized women tend to be lighter than the infants of taller women.

The yield of milk, both in volume and in solids, tends to be lower in the undersized women.

There may be a tendency for the concentration of fat in the milk of the undersized women to be higher than in the taller women.

Variations of the state of nutrition of women, within normal limits, do not influence the weight of their infants or the weight of solids secreted in their milk.

The concentration of solids not fat is definitely lower in the milk of thin women than in that of better nourished women. There may also be a tendency for the concentration of fat to be lower in the milk of the thin women.

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THE TREATMENT OF RUPTURED TUBAL GESTATION.

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THE question of delayed *versus* immediate operation in ruptured tubal gestation has formed a subject of debate amongst gynæcological surgeons for many years. Recently I was permitted to hear two surgeons of this city strongly advocating the delayed operation.

I investigated the records of the cases at Sydney Hospital over the last ten years and, together with the study of the available literature and my own experience, I am definitely of the opinion that immediate operation gives the patient the best chance of recovery.

Table I shows twenty-one cases from the records of Sydney Hospital, all of which are cases of definite tubal rupture; in all cases free blood was present in the peritoneal cavity and the patients showed symptoms of severe collapse. Leaking ectopic pregnancies and tubal abortions with small amounts of blood and only minor degrees of collapse have not been included, since immediate operation in these cases is not of such vital importance, but should, in my opinion, be done all the same.

Nineteen of these twenty-one reported patients underwent immediate operation and eighteen were discharged from hospital alive and well; one died suddenly seven days after operation from some unknown condition, death being preceded by paralysis of the legs. On the day of her death this patient's temperature was 36·9° C. (98·4° F.) and pulse rate 90, and her general condition was apparently quite good.

Two patients were not operated on and both died. One was in hospital for twelve hours and received treatment for her collapse; the other was in hospital for only two hours, and as she was *in extremis*, it was thought inadvisable to operate. In both cases a ruptured tubal gestation with free blood in the peritoneal cavity was found at autopsy.

In Cases XIV and XVIII the patients were in extreme collapse and I could with difficulty feel an extremely rapid and feeble pulse in the radial artery. In both instances a rapid operation, in one case under gas and oxygen anæsthesia combined with autotransfusion, converted women who were *in extremis* to eventual normal health.

In the discussions in the available literature the majority of the opinions are in favour of operation as soon as possible. Congdon,[1] at a meeting of the American Association of Obstetricians and Gynæcologists, quotes Schauta, who, after a careful study of the literature, collected 123 operative cases with a mortality of 5·7%, and 121 cases treated palliatively with a mortality of 86·89%.

Stoddard[2] states that he has never seen a patient with a ruptured ectopic gestation recover without operation, but this is not always the case, for most gynæcologists have discovered at some time or another in the pouch of Douglas a mass of old blood clot containing a dead embryo. I contend, however, that the majority of these cases have been tubal abortions or ruptures with only slight blood loss and minor degrees of collapse.

Hawks,[3] in reviewing 184 cases of ruptured ectopic pregnancy among patients who were seen in three New York hospitals and who were in critical condition, found that of 113 operated on immediately, ten died (8·8%). Five only of these deaths were due to post-operative shock. Of 71 patients treated expectantly, with deferred operations, 12 died (17%). Seven of these twelve died of hæmorrhage before operation was performed.

TABLE I.
Cases of Tubal Rupture from the Records of Sydney Hospital During the last Ten Years.

Case Number.	Patient.	Age.	Pulse Before Operation.	Temperature Before Operation.	Operation.	After Treatment.	Remarks.
1	A.L.	33	140	35·8° C. (96·4° F.)	Salpingectomy.	Rectal saline injections and morphine.	Uninterrupted recovery.
2	E.G.	29	135	36·2° C. (97·2° F.)	Salpingectomy.	Rectal saline injections and morphine.	Uninterrupted recovery.
3	H.M.	25	110	36·1° C. (97·0° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
4	A.L.	21	140	35·8° C. (96·4° F.)	Salpingectomy.	Glucose and rectal saline injections and morphine.	Uninterrupted recovery.
5	D.W.	24	130	36·5° C. (97·8° F.)	Salpingectomy.	Stimulants and morphine.	Uninterrupted recovery.
6	M.S.	30	150	36·1° C. (97·0° F.)	Salpingectomy.	Stimulants, rectal saline injections and morphine.	Uninterrupted recovery.
7	M.L.	32	130	37·3° C. (99·2° F.)	Salpingectomy.	Saline, rectal injections and pituitrin.	In this case rupture occurred while the patient was in hospital
8	N.T.	32	110	37·5° C. (99·6° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
9	I.M.	27	100	35·6° C. (96·0° F.)	Salpingectomy.	Coffee and brandy enema, morphine.	Uninterrupted recovery.
10	A.M.	34	114	36·9° C. (98·4° F.)	Salpingectomy.	Rectal saline injections and morphine.	Uninterrupted recovery.
11	F.L.	32	130	37·1° C. (98·8° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
12	D.C.	26	116	36·5° C. (97·8° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
13	M.S.	23	120	36·7° C. (98·0° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
14	M.S.	29	?	?	Salpingectomy.	Intravenous saline injections during operation. Glucose and saline rectal injections, morphine and pituitrin.	Pulse imperceptible before operation. Recovered.
15	F.G.	29	140	36·3° C. (97·4° F.)	Salpingectomy.	Saline rectal injections and morphine.	Uninterrupted recovery.
16	S.W.	38	130	36·5° C. (97·8° F.)	Salpingectomy.	Morphine.	Uninterrupted recovery.
17	E.S.	41	130	36·1° C. (97·0° F.)	Salpingectomy.	Saline solution in abdomen. Intravenous injections of saline and glucose and saline rectal injections and morphine.	Uninterrupted recovery.
18	T.C.	23	?	36·1° C. (97·0° F.)	Salpingectomy.	Autotransfusion, morphine.	Pulse imperceptible. Patient *in extremis*, recovered.
19	R.F.	30	130	36·1° C. (97·0° F.)	Salpingectomy.	Transfusion; intravenous and subcutaneous saline injections.	This patient was quite well for seven days, when she developed paralysis of lower limbs and died suddenly. At time of death pulse was 90 and temperature normal. Cause of death unknown.
20	G.S.	39	120	36·1° C. (97·0° F.)	None.	Given transfusion and watched. Treated for shock.	Died twelve hours after admission; autopsy revealed ruptured ectopic gestation, free blood in abdomen.
21	M.C.	27	?	?	None.	Stimulants *et cetera*.	Patient was *in extremis* and died two hours after admission. Autopsy: ruptured tubal gestation and free blood.

Carl Henry Davis, discussing the waiting policy in his latest book, writes as follows:

> Many women do react favourably under this waiting policy, but for those who do not many valuable hours and more precious blood have been wasted. This method is all right for those who do react, but very dangerous and often fatal for those who do not.

Litzenberg writes in Nelson's "Surgery":

> Even though there be collapse, shock and alarming blood loss, an operation should be done as promptly as circumstances will permit; the blood flow must be stilled. Until the pumping artery is tied, there is no safety.

The collapse, or so-called "shock", of these patients is due to the loss of blood, and a blood transfusion will cause a sudden dramatic improvement in the patient's condition. However, unless the transfusion is followed immediately by operation and ligation of the bleeding vessel, the patient will quickly lapse into her former critical condition.

The operation should be performed as rapidly as possible, with due regard to efficiency; the presenting blood clots and free blood obscuring the route to the tube should be quickly removed, but no time should be wasted looking for clots or mopping up blood in the various *culs-de-sac* of the peritoneal cavity. The *per vaginam* examination done before operation will inform the surgeon which tube is affected, and he can go straight for this tube on opening the peritoneal cavity.

When the patient is *in extremis* a blood transfusion is of great value, but the operation should not be delayed on that account. The coagulation test for compatibility takes very few minutes to

perform and these preliminaries can be done simultaneously with preparations for operation. The transfusion is then given before or simultaneously with the operation, but the operation should not be held up by the transfusion. If no immediate donor is procurable, then autotransfusion or saline solution given intravenously with 6% glucose is of benefit, but neither is of such great value as the blood from a suitable donor.

After operation the patient should be kept warm and flat in bed and under morphine; the foot or head should not be raised, as is often done. It is remarkable how quickly these patients progress after the initial operative shock has disappeared.

In remote country districts, where rapid, smooth transport is not available, or where quick surgery is not possible, or where the medical attendant doubts his ability to deal with the condition rapidly and effectively, then the "waiting policy" will have to be adopted in critical cases. Under city and suburban conditions, however, I can see no advantages, but many disadvantages, in the dangerous "waiting policy".

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(1) C. Congdon: *Surgery, Gynecology and Obstetrics*, Volume XI, 1910, page 520.
(2) T. A. Stoddard: *Surgery, Gynecology and Obstetrics*, Volume IX, 1908, page 484.
(3) E. M. Hawks: "Immediate Versus Delayed Operation in Cases of Collapse following Ruptured Ectopic Pregnancy", *Surgery, Gynecology and Obstetrics*, Volume XXXVI, February, 1923, page 232.

RHEUMATIC HEART LESIONS IN 3,000 AUSTRALIAN POST MORTEM EXAMINATIONS.[1]

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THE results here set out are those found in 2,700 *post mortem* examinations at the Adelaide Hospital and 300 at the Mental Hospital at Parkside. The lesions met with in these autopsies have all been tabulated (see Tables I and II) and these tabulated results are now analysed from the point of view of the effects of acute rheumatic fever on the heart.

There were 88 cases (2·9%) of valvular disease which were certainly or probably due to acute rheumatic fever; in 17 of these cases the surfaces of the pericardium were adherent. There were 28 other cases of *synechia pericardii* not associated with valvular lesions; it is probable that many of these 28 cases had their origin in acute rheumatic fever. If we accept them all as such, the total rheumatic heart cases would amount to 116, or 3·9%. During the same time there were 23 cases of syphilitic disease of the aortic valve and 66 examples of malignant endocarditis (18 of these being included under the rheumatic fever cases as having clearly a rheumatic basis). There were also 75 examples of lesions in the valves of a nondescript nature, this number including 10 examples of calcified knobby aortic cusps and 7 other cases which may have had a rheumatic basis. The rheumatic cases presumably included under this nondescript section probably compensate for the examples of adherent pericardium which had not a rheumatic basis. It is probable, therefore, that about 116 cases in the 3,000 showed lesions in the heart or pericardium as the result of previous acute rheumatic fever.

In the 88 cases of valvular disease the mitral valve was affected in 76, the aortic valve in 39, the tricuspid valve in 8, and the pericardium was adherent in 28.

At the same time there were 142 hypertrophied and dilated hearts in which the hypertrophy and dilatation was not due to valvular disease or pulmonary lesions; 66 of these were associated with renal fibrosis and 76 showed little or no renal fibrosis and were presumably examples of essential hypertension.

[1] This paper consists in part of a contribution to the discussion of the paper by Dr. Guy Lendon published in this journal on May 12, 1934.

TABLE I.
Heart Lesions in 3,000 Post Mortem Examinations (Adelaide Hospital 2,700, Mental Hospital 300).

Lesion.	Number.	
Rheumatic valvular disease	88	143
Rheumatic valvular disease (doubtful)	7	
Malignant endocarditis (18 included under rheumatic)	66	
Syphilitic disease of the aortic valve	23	
Calcified knobby aortic cusps with stenosis	10	
Hypertrophied and dilated hearts with renal sclerosis	66	142
Hypertrophied and dilated hearts without adequate renal cause, presumably essential hypertension	76	

TABLE II.
Distribution of Lesions of Rheumatic Heart Disease in 3,000 Post Mortem Examinations.[1]

Distribution.	Number.
Mitral stenosis	14
Mitral stenosis and malignant endocarditis	5
Verrucosities on mitral cusps	7
Mitral stenosis and *synechia pericardii*	8
Mitral stenosis and recent pericarditis	1
Mitral regurgitation	8
Mitral and aortic disease, mostly stenoses	15
Mitral and aortic disease, with malignant endocarditis	6
Mitral and aortic disease with *synechia pericardii*	5
Mitral and tricuspid disease	4
Mitral and tricuspid disease with malignant endocarditis	2
Mitral, aortic and tricuspid disease	2
Aortic disease	4
Aortic disease with malignant endocarditis	4
Aortic disease with *synechia pericardii*	3
Synechia pericardii only (probably not in all cases rheumatic)	28

[1] In the 88 cases of rheumatic valvular disease the mitral valve was affected in 76; the aortic valve was affected in 39; the tricuspid valve was affected in 8; and the pericardium was adherent in 16.

Mitral Stenosis.[1]

There were fourteen cases of mitral stenosis; eleven occurred in females and three in males.

With recent vegetations, F.21, F.28, F.45. With stenosis alone, F.27, F.37, F.45, F.47, F.48, F.58, F.67 (some stenosis), F.71, M.38, M.48 and M.68.

Mitral Stenosis and Malignant Endocarditis.

There were five cases of mitral stenosis and malignant endocarditis, one in a female and four in males.

F.36, M.19, M.23, M.32 and M.61.

[1] In the description of cases in this article F. = female, M. = male; the age is stated in years immediately after the letter M. or F.

Verrucosities on the Mitral Valve.

There were seven cases with verrucosities on the mitral valve, all in females.

F.21, F.21 (firm, yellowish, pearly rheumatic vegetations on the mitral cusps), F.22 (fine warty vegetations), F.28 (verrucose vegetations, ? rheumatic), F.49 (old vegetations), F.53 and F.86.

Mitral Stenosis with Synechia Pericardii.

There were eight cases of mitral stenosis with *synechia pericardii*, four in females and four in males.

F.14 (slight thickening of mitral cusps), F.21 (early stenosis), F.66, F.18 (with polypoid malignant endocarditis), M.29, M.40, M.69 (partial pericardial adhesions), M.36 (aboriginal, fibrosis of mitral valve with mural endocarditis).

Mitral Stenosis with Recent Pericarditis.

There was one case of mitral stenosis with recent pericarditis.

F.63 (recent slight pericarditis).

Mitral Regurgitation.

There were eight cases of mitral regurgitation, four in females and four in males.

Autopsy number 83/25: F. 23. The mitral valve was thickened and fibrotic, no stenosis was present. The left auricle was very dilated with endocarditis of its wall and of the mitral valve. The aortic valve was normal, the tricuspid valve was a little thickened. The left ventricle was hypertrophied. Acute heart failure had occurred.

Autopsy number 43/23: F.30. Slight but definite thickening of the mitral cusps was present and slight shortening; the edges were somewhat rounded and sharply defined. The left ventricle was hypertrophied with *ante mortem* thrombi.

Autopsy number 208/26: F.44. Thickening and distorsion of mitral cusps were present without stenosis.

Autopsy number 51/22: F.61. The mitral orifice admitted three fingers; it was apparently incompetent; the cusps were thickened towards the edges and were nodular. The *chordæ tendineæ* were thickened and shortened. All parts of the heart were hypertrophied; the weight of the heart was 622·6 grammes (22 ounces).

Autopsy number 50/23: M.57. The mitral valve was distinctly thicker than normal, with the free edges rounded and the surface presenting a yellowish appearance; the aortic cusp had a yellowish opacity. The *columnæ carneæ* were prominent. The left ventricle was hypertrophied and dilated; the right side of the heart was dilated.

Autopsy number 101/22: M.61. The mitral orifice admitted three fingers; the cusps were thickened and somewhat contracted. The *chordæ tendineæ* were markedly thickened. The aortic valve was unaffected. Acute heart failure had occurred.

Autopsy number 55/27: M.70. The mitral orifice admitted three fingers; the cusps were much thickened. The heart was hypertrophied.

Autopsy number 189/30: M.71. The mitral orifice admitted three fingers; the cusps were thickened and sclerosed, in places calcified. The heart was hypertrophied, its weight was 721·6 grammes (25½ ounces). Auricular fibrillation had occurred.

Mitral and Aortic Disease.

The following were cases of mitral and aortic disease.

Autopsy number 28/28: F.13. Thickening of mitral valve and recent vegetations on the aortic valve were present.

Autopsy number 124/30: F.21. Early mitral stenosis was present. There was slight thickening of the aortic cusps.

Autopsy number 172/25: F.24. Aortic disease was commencing.

Autopsy number 124/32: F.31. Mitral and aortic stenosis were present.

Autopsy number 205/24: F.62.

Autopsy number 179/30: F.68. The mitral orifice admitted two fingers. There were some calcification and puckering of the mitral cusps. Some calcification was present in the aortic cusps. There was a rheumatic history.

Autopsy number 32/23: M.30. Old and recent vegetations were present.

Autopsy number 12/31: M.34. Sclerosis of the mitral and aortic valves was present with stenosis and calcification.

Autopsy number 103/24: M.44. Mitral stenosis was present with calcification, and aortic disease with calcification.

Autopsy number 23/33: M.47. Some mitral stenosis and thickening of the aortic cusps were present.

Autopsy number 202/23: M.49. Old rheumatic mitral and aortic endocarditis was present with calcification.

Autopsy number 7/27: M.49. Mitral and aortic stenosis was present with calcification.

Autopsy number 183/31: M.55. Aortic stenosis was present with calcification; calcification was present in the mitral cusps. The hypertrophied and dilated heart weighed 728·7 grammes (25¾ ounces). There was a rheumatic fever history.

Autopsy number 5/28: M.55. Acute vegetations were present on the aortic valve.

Autopsy number 28/31: M.63. Mitral stenosis was present with slight adhesion between two aortic cusps.

Mitral and Aortic Disease with Malignant Endocarditis.

The following were cases of mitral and aortic disease with malignant endocarditis.

Autopsy number 187/25: F.41.

Autopsy number 24/26: F.49. The aortic cusps were thickened, malignant endocarditis of the mitral valve was present.

Autopsy number 200/27: M.18. Old rheumatic vegetations were present on the mitral cusps. Sclerosis of the aortic cusps was present. Malignant endocarditis of both mitral and aortic valves was present.

Autopsy number 53/26: M.20.

Autopsy number 68/32: M.20. Polypoid malignant endocarditis on old rheumatic disease was present.

Autopsy number 163/30: M.50. Mitral stenosis and sclerosis of the aortic cusps were present.

Mitral and Aortic Disease with Synechia Pericardii.

The following were cases of mitral and aortic disease with *synechia pericardii*.

Autopsy number 90/26: F.12. Old fibrosis of the mitral valve was present. There were recent vegetations on the mitral and aortic valves.

Autopsy number 28/28: F.13. Old thickening of the mitral valve was present. There were recent rheumatic vegetations on the aortic valve.

Autopsy number 149/26: F.15. Recent lesions of both valves with old endocarditis of the mitral valve were present.

Autopsy number 120/33: F.36. Vegetations were present on both valves. There was a rheumatic fever history.

Autopsy number 70/28: M.26. Mitral stenosis was present with slight aortic fibrosis.

Mitral and Tricuspid Disease.

The following were cases of mitral and tricuspid disease.

F.25 (chronic, with regurgitation), F.30, F.72, M.76 (thickening with irregular contraction, doubtfully rheumatic).

Mitral and Tricuspid Disease with Malignant Endocarditis.

The following were cases of mitral and tricuspid disease with malignant endocarditis.

F.19, M.22.

Mitral, Aortic and Tricuspid Disease.

The following were cases of mitral, aortic and tricuspid disease.

F.47, stenoses of the three valves. M.42, slight thickening of the mitral and aortic cusps with sago-grain vegetations on the tricuspid.

Aortic Disease.

The following were cases of aortic disease.

Autopsy number 188/26: M.25. Retraction and calcification were present.

Autopsy number 98/20: M.34. Regurgitation with mitral incompetence was present.

Autopsy number 44: M.42. Thickening of aortic cusps with nodular surface was present.

? Autopsy number 130/22: M.80. Aortic stenosis was present.

Aortic Disease with Malignant Endocarditis.

The following were cases of aortic disease with malignant endocarditis.

Autopsy number 90/27: M.22. Stenosis was present.

Autopsy number 123/25: M.42. Stenosis was present.

Autopsy number 66/25: M.45. Fibrosis and calcification were present.

Autopsy number 186/24: M.53. Stenosis and calcification were present.

Aortic Disease and Synechia Pericardii.

The following were cases of aortic disease and *synechia pericardii*.

Autopsy number 112/25: M.54. Stenosis and regurgitation were present.

Autopsy number 138/27: M.59. Stenosis with calcification was present.

Autopsy number 141/21: M.70. The aortic cusps were calcified.

Synechia Pericardii Only.

In the following cases *synechia pericardii* only was present.

F.20, F.27, F.31, F.35 (?), F.37, F.46, F.47 (with syphilitic aortitis), F.49, F.60, F.64, F.82, M.22, M.37, M.40, M.51, M.53, M.56 (with syphilitic aortitis), M.57, M.60, M.60, M.64 (with hypertrophied and dilated heart), M.65, M.70, M.71, M.76, M.77, M.79, M.90.

Eleven of these were in women, five of whom were under the age of forty years, and in one the condition was associated with syphilitic aortitis. Seventeen were in men, of whom only two were under forty years, and one patient had also syphilitic aortitis.

Heart Lesions with a Possible Rheumatic Basis.

Amongst the 75 cases in which nondescript lesions of the valves were present, the following 17 more particularly may have had a rheumatic basis.

Calcified Knobby Aortic Cusps giving rise to Stenosis.

Ten cases: F.69, M.40, M.41, M.55, M.56, M.58, M.59 (three cases), M.62.

Other Cases.

Autopsy number 176/30: M.17. Thickening and puckering of the mitral valve were present.

Autopsy number 169/27: M.21. Some sclerosis of the mitral valve was present.

Autopsy number 63/29: M.32. The aortic and mitral valves were contracted, calcified and irregular, with stenosis.

Autopsy number 115/29: M.35. Two aortic cusps were fused at their edges and their common attachment was calcified and lengthened.

Autopsy number 195/30: F.37. Puckering of the mitral cusps was present with old vegetations.

Autopsy number 205/25: M.48. Calcification of the aortic cusps was present with stenosis.

Autopsy number 141/24: M.68. Calcification and deformity of the aortic and mitral cusps were present.

Discussion.

In considering the preceding lesions it should be remembered that the patients, however, have not necessarily died as the result of the rheumatic condition. Most of the younger patients did die from the valvular damage, but in the older patients the valvular difficulty may be successfully overcome for years and they may eventually die from something else, although with increasing years the valvular damage may have degenerative changes superadded and eventually interfere with the competence of the heart's action.

Sex Incidence.

There seem to be some interesting associations between different lesions and sex. Thus mitral stenosis seems definitely commoner in women (11) than in men (3), though when malignant endocarditis is superadded we found that only one of our cases occurred in a woman and four in men.

The whole of the seven persons recorded as having verrucosities on the mitral valve were all women. Mitral stenosis associated with *synechia pericardii* shows an equality in the sexes, namely, four of each. The association of mitral with tricuspid disease was seen in four women and two men. Aortic disease, with or without malignant endocarditis and with or without *synechia pericardii*, affected males only, eleven in all. Mitral and aortic disease affected thirteen males and eight females, but when these valves were affected and the pericardium was adherent there were four females and only one male.

These results, though small, seem to show again a greater tendency in rheumatic disease for the mitral valve to be affected in women than in men, and a greater liability for the aortic valve to be affected in men. This perhaps means that in men who are frequently engaged in hard work there is a greater strain on the aortic cusps, although even then we do not get any explanation why disease of the mitral valve is so much commoner in women than disease of the aortic valve.

Ages of the Patients.

There seems no doubt of the correctness of the diagnosis of rheumatic heart disease in the younger patients. In those over fifty, however, doubt sometimes arises and it may be hard to decide whether atheromatous changes and calcification have occurred in a valve which has undergone fibrosis as the result of rheumatic fever, or whether these degenerative changes have no such basis. It will be noted that

we have placed under the heading "Heart Lesions with a Possible Rheumatic Basis" those cases of calcified knobby aortic cusps which sometimes occur in patients well under fifty, as well as those occurring in persons who are older. There are three possible explanations of these cases. One is that they represent degenerative changes superadded to rheumatic fibrosis; the second, that they are purely degenerative; and the third, that they may represent the aftermath of low-grade infective endocarditis from which there has been a recovery. At present I have an open mind on this question, but their occurrence from time to time in relatively young persons and the absence of much atheroma elsewhere in other cases would seem to exclude a purely degenerative basis for them.

Mitral Regurgitation.

Pure mitral regurgitation due to rheumatic disease seems to be a comparatively rare condition. In the 76 examples in which the mitral valve was diseased there were approximately 61 examples of mitral stenosis and only eight examples of mitral regurgitation without stenosis. In these eight examples the cusps were thickened and retracted, with the free edge usually rounded and the *chordæ tendineæ* thickened and shortened. The notes do not state whether there were any examples of mitral regurgitation due to rheumatic disease in which the *chordæ tendineæ* were not contracted but, contrariwise, lengthened. But the latter condition, if present, would probably have been noted.

Systolic Cardiac Murmurs.

In 300 *post mortem* examinations at the Adelaide Hospital and the Mental Hospital at Parkside there were approximately 143 cases of rheumatic valvular disease and/or malignant endocarditis. In a considerable number of these cases a malignant endocarditis affected the mitral valve and might have caused a regurgitant as well as an obstructive murmur. About 61 of the rheumatic cases were examples of mitral stenosis. In only eight of the rheumatic cases was regurgitation present without stenosis. There were 23 examples of syphilitic disease of the aortic valve which would cause a regurgitant murmur when eventually the left ventricle dilated. The same applies to 10 examples of calcified knobby aortic cusps with stenosis. There were 66 examples of hypertrophied and dilated hearts with renal sclerosis, and 76 examples of hypertrophied and dilated hearts without adequate valvular or renal cause and presumably due to essential hypertension. These 142 examples of hypertrophied and dilated hearts not due to valvular disease would presumably all have given rise to regurgitant murmurs at the mitral orifice.

It will thus be seen that eight cases of mitral regurgitation due to rheumatic disease would be expected to give rise to a systolic murmur at the apex; that 23 examples of syphilitic disease of the aortic valve, 10 examples of calcified knobby aortic cusps with stenosis, and 142 examples of hypertrophied hearts with high blood pressure would all, when the left ventricle failed and dilated, give rise to, presumably, systolic murmurs at the apex. Most of the 61 cases of mitral stenosis would probably, from the rigidity of the cusps, allow a certain amount of regurgitation to take place.

THE CONSERVATIVE TREATMENT OF FACIAL PARALYSIS.

By N. D. ROYLE, M.D., Ch.M. (Sydney), F.R.A.C.S.,
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FACIAL paralysis is the commonest peripheral nerve lesion. In my series of thirty-four cases the onset was sudden in twenty and usually followed exposure to cold in some form. Of the remaining fourteen, four occurred in conjunction with other sequelæ of poliomyelitis, one followed encephalitis, four resulted from accidental trauma, and five were associated with mastoiditis.

Neuritis of the facial nerve or of the geniculate ganglion is by far the commonest cause of the lesion, and the paralysis must be attributed to interruption of conductions of the facial nerve by œdema in the facial canal. The results of consequent treatment will bear out this idea.

Prognosis in a Case of Facial Paralysis.

The facial nerve is a mixed nerve and, in addition to supplying the superficial musculature of the face, subserves deep sensibility. Thus a means of prognosis is available. If deep sensibility is absent, a complete lesion of the facial nerve may be diagnosed in both traumatic and inflammatory cases. If hyperæsthesia or normal sensation is present on the side of the paralysis, an incomplete lesion may be diagnosed and a hopeful prognosis of immediate recovery may be given. In recovery any mixed nerve is hyperæsthetic, so that if sensitiveness appears in the deep sensation of the face early recovery may be expected. Testing for deep sensibility is therefore more important than carrying out electrical tests, which simply indicate whether the muscles are denervated or not. Reaction of degeneration does not indicate inability to recover.

Treatment.

Massage.

Since facial paralysis is most commonly the result of œdema in the facial canal, treatment should be directed towards the relief of that œdema. The absorption of lymph is mainly through the lymphatic vessels, and the plan of treatment is to empty the lymphatic vessels draining the mastoid area. This is done by massaging the mastoid area and the side of the neck from the occiput to the chin on the affected side. The muscles of the face must not be massaged. The facial muscles are slender and attached to the skin, and vigorous handling elongates their fibrous tissue. This elongation, I am certain, is the cause of incomplete recovery when the nerve supply has returned. Patients are often seen months or even years after

nerve regeneration with weak facial movements due to the altered lengths of muscle occasioned by vigorous massage. This applies to normal faces also. When the muscles are denervated they are very vulnerable, but when the nerve supply has returned gentle stroking movements may be given.

Rest.

One of the most important aspects of treatment in a denervated or recovering muscle is to prevent constant elongation by gravity. In the face this

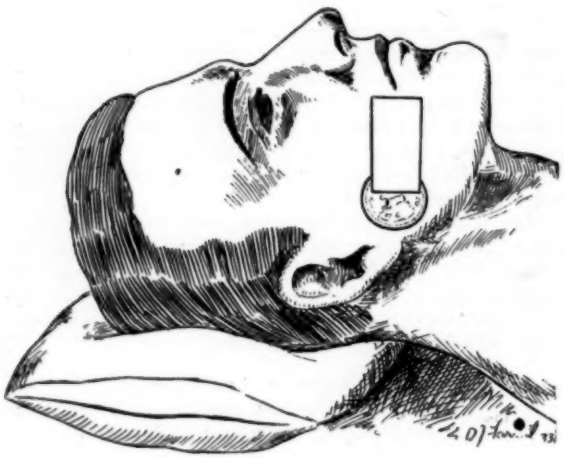

FIGURE I.

is best accomplished by adhesive plaster. Wire splints from the angle of the mouth to the ear rest only the buccal muscles, whereas strapping from the angle of the mouth to the forehead rests practically all the muscles of the mouth and eye and maintains the position of the face against the action of gravity. Care should be taken to place the strapping in a lateral position on the forehead, so that it does not pull on the frontalis muscle. Rest is a very important feature of treatment, and I have seen movements return to a face from which they have been absent for months, after a few days' rest, which allows the muscles to take up their slack. This result, of course, appears only when the nerve has recovered.

Electrical Treatment.

It is constantly stated in medical literature that electrical treatment keeps the facial nerves in tone, but denervated muscles have no tone and tone cannot be induced in them by making them contract. Denervated muscles are in a state of constant contraction and the wasting is really due to fibrillation (Langley). Of what use, then, is contraction by the galvanic current? If it does anything, it increases the rate of wasting. The faradic current should be mentioned only to be condemned, since it has no effect on denervated muscles; and when the muscles have recovered, active movement is the most appropriate treatment. The galvanic current has no effect on the rate of recovery of nerve, and active movements return to the face long before faradic stimulation is possible. To apply electrical treatment to the face in any stage is a waste of time and money.

Active Movements.

In the treatment of facial paralysis active movements are second in importance only to draining of the mastoid area. The average time of recovery in an acute case is about ten days, and in this short period the wasting is about 20% only. In a well managed case movement never really leaves the face and consequently denervation is not complete and the wasting is negligible. In both instances the movements of the face are normal when the nerve has recovered, but if the denervation has been long continued, wasting and the inaccurate movement due to disuse render reeducation necessary. For example, very few individuals have complete control of one side of the face, and if asked to move the defective side, patients commonly use the normal side. This must be prevented if the facial muscles on the defective side are to be tested properly,

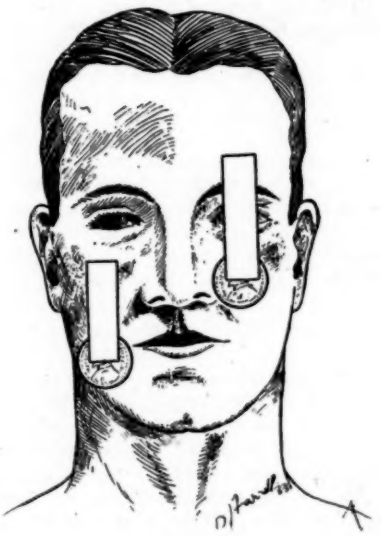

FIGURE II.

because in such an act as smiling the movements of the normal side obscure the smaller movements of the defective side by drawing the face in the normal direction. To test the muscles used in smiling the normal side should be held passively in the position of relaxation, while the patient is instructed to stop smiling with the normal side and to endeavour to do a small movement with the affected side. This minimization of effort is very important in reeducation and in testing defective muscles. In making a big mental effort the patient will unconsciously substitute other similar movements for the required one. Accurate innervation is not consistent with great mental effort.

In testing a face, movement may often be first seen in the *orbicularis palpebri* and may be detected by raising the face passively on the defective side and instructing the patient to close the eye. After movement has appeared, the next step is to restore it to normal. As stated previously in this paper, this is very difficult if the face has been massaged vigorously, because if facial muscles have been elongated, they have consequently too much slack to take up. All muscles have slack to be taken up when movement is started from the relaxed position. Since the facial muscles are muscles of expression and are associated with the interpretation of conscious states, they are normally practically continuously innervated. In addition, they support the face against gravity and this tends to elongate them. This stretching, as in all muscles, induces tone.

How to Exercise Defective Facial Muscles.

Muscles will not recover full power unless a physiological demand is made on them for an increase in structure. Facial muscles, as all other muscles, must have gradually increasing resistance applied to them for the physiological demand to be made. Fatigue does not damage a recovering muscle provided it is not prolonged and is followed by a period of rest. It creates the normal demand for an increase in structure.

How to Load the Facial Muscles.

Loading of the facial muscles may be done by means of "ZO" strapping and coins. Thus for the occipito-frontalis a piece of strapping is attached to the forehead just above the eyebrow and a coin, a threepence, sixpence, shilling or two-shilling piece, stuck on to the lower end to act as loading; as the function improves, coins of larger size are used. Similarly a coin may be attached to the skin with strapping in the region of the malar bone to make loading for the muscles of the eye. The accompanying drawings illustrate the method. When the muscles have recovered, gentle massage may be used after exercise.

Reports of Cases.

CONGENITAL ANEURYSM OF THE LEFT ANTERIOR CEREBRAL ARTERY.

By A. A. MERRITT, M.B., B.S.,

AND

M. STARICOFF, M.B., B.S.,

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DR. JEREMY'S discussion of subarachnoid hæmorrhage and reports of his cases in THE MEDICAL JOURNAL OF AUSTRALIA of October 21, 1933, in which he states that the prognosis is doubtful, has led us to record this particular case. Fernside, quoted in "Recent Advances in Neurology", has recorded a case in which the interval between two hæmorrhages was twenty-two years. This present case shows an interval of seventeen years between the patient's first hæmorrhage and the subsequent hæmorrhages which caused his death. During that seventeen years the patient was almost entirely free from symptoms.

Clinical History.

H.H., a male, aged forty-nine years, a labourer, was admitted to the Perth Hospital on October 10, 1933. He complained that during the last seventeen years he had had bouts of headache lasting a few hours, occurring very infrequently, unassociated with any other symptoms. Ten days before admission, whilst resting after some fairly light work, he suddenly noticed a pain which shot through his head in the temporal region, but did not radiate down the neck. Within a minute of the onset of the pain he became unconscious and remained so for a quarter of an hour.

Immediately on regaining consciousness he became nauseated and vomited. The physician under whom he placed himself found "blood-stained cerebro-spinal fluid under increased pressure" and sent the patient to Perth. On arrival he volunteered that since the onset of his illness he had had bitemporal headache, vomiting and weakness. Further questioning elicited the fact that for some time he had had dimness of vision, and in the last few days had seen "specks before the eyes". He had never at any time had diplopia. Between the spasms of headache he complained of visual hallucinations. The only illnesses he had had previously were gonorrhœa and trench fever at the war, and in 1916 he was confined to a military hospital in Malta with exactly the same symptoms of which he now complained.

That his symptoms were the same then as now was beyond question. Between then and now he had had slight attacks of headache.

Examination revealed that the radial artery was slightly thickened, the systolic blood pressure was 112, and the diastolic 78 millimetres of mercury. The temperature was 36·8° C. (98·2° F.), the pulse rate was 72, and the respirations numbered 28 in the minute. Urinary examination revealed a specific gravity of 1020. No albumin was found. Microscopically the urine was clear. Examination of the heart, lungs and abdomen revealed nothing of importance. The pupils reacted poorly to light, but were equal. The right upper superficial abdominal reflex was absent and the left plantar reflex was equivocal, the right flexor. No sensory changes were observed and, apart from the reflex changes, examination physically revealed no abnormality except a mild degree of bilateral optic neuritis.

Lumbar puncture on admission yielded a yellow fluid under a pressure of 220 millimetres of water. Chemical examination revealed that the chlorides were 670 milligrammes per 100 cubic centimetres and the total protein 180 milligrammes per 100 cubic centimetres. Microscopy revealed 500 cells, mostly lymphocytes, per 100 cubic millimetres. The Wassermann test in the cerebro-spinal fluid gave no reaction.

To determine whether there was any bone erosion of the base of the skull an X ray examination was made and revealed nothing.

The convalescence was uneventful till just six days after admission, when severe headache was complained of. The patient vomited and neck stiffness was found. Lumbar puncture revealed grossly blood-stained fluid.

He gradually improved until three days later (October 19, 1933), when he suddenly became cyanosed, sweated and frothed at the mouth. The pulse rate, which hitherto had fluctuated between 60 and 80, suddenly rose to 160 and the pulse became "softer". Clonic contractions of both hands set in, more marked in the left hand. Markedly increased neck stiffness was noticed. Lumbar puncture again revealed grossly blood-stained fluid under increased pressure. The next day the patient could not speak.

Lumbar puncture was again repeated on November 23, 1933, and yielded blood-stained fluid with a pressure of 168 millimetres of water.

From this time onwards the patient gradually sank deep into coma, the pulse rate remained rapid and the patient died on October 29, 1933.

The temperature variations throughout the course of the disease were interesting. From October 10 to 16 the temperature showed a slight nightly elevation which never

exceeded 37·8° C. (100° F.). On October 16, 1933, he had a hæmorrhage and the temperature on the following day was 38·3° C. (101° F.), but fell within forty-eight hours to normal and rose again on the evening of October 19 to 38·3° C. (101° F.) again, coinciding with a fresh hæmorrhage, as proved by lumbar puncture. The temperature fell again within forty-eight hours to normal, but rose again on October 20, and from then onwards till death. The chart showed an evening fluctuation of 38·3° C. (101° F.).

Post Mortem Findings.

Permission was granted for a limited *post mortem* examination, which revealed a large amount of blood in the subarachnoid space, over the cerebrum, and around the base of the brain in the region of the optic chiasma. Dissection revealed an aneurysm, about one centimetre in diameter, of the left anterior cerebral artery, at its junction with the anterior communicating artery. The aneurysm showed a small area through which hæmorrhage had taken place, ploughing up the right frontal lobe, which contained a cavity full of new blood clot. That this cavity communicated with the ventricles was not proved, because it was desired to preserve the specimen without inflicting too much damage upon it. No other aneurysm was found.

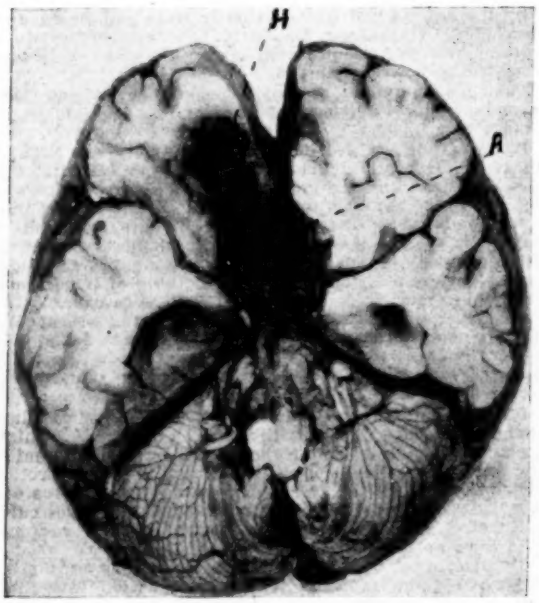

FIGURE I.

Photograph showing the lesion. The aneurysm, one centimetre in diameter, is shown on the left anterior cerebral artery—"A". The right frontal lobe shows a large area which is occupied by recent blood clot—"H". The optic nerves, chiasma and infundibulum, together with portion of the frontal and temporal lobes, have been removed and the brain underlying the circle of Willis cut away in order to demonstrate the circle of Willis and its branches and to show the track of the blood infiltrating the frontal lobe from the aneurysm.

Conclusion.

A case of congenital aneurysm of the anterior cerebral artery is reported which demonstrated that a patient may have a considerable time (seventeen years in this case) between the first hæmorrhage and a succeeding one.

That it is a congenital aneurysm is fairly well established, since the first hæmorrhage occurred when the patient was aged thirty-two, and the cerebro-spinal fluid did not react to the Wassermann test. Besides this, the aneurysm occurred at one of the commonest sites of congenital aneurysms, and no other ætiological factor was discovered.

Acknowledgement.

We are grateful to Dr. Streich, Honorary Physician to the Out-Patients, Perth Hospital, for giving us permission to record this case.

ARTHROKATADYSIS OF THE HIP JOINT.

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THE patient was an elderly woman, married, aged seventy years, who came to the out-patient department of the Royal North Shore Hospital complaining of severe pain in her left thigh for the previous fourteen years and in the right thigh during the previous six months; at that time (November, 1933) the pain on the right side was more severe than on the left. There had never at any time been pain in either hip; but occasionally there had been backache, low down, and some pain in the knee.

In December, 1933, both hips were equally affected and so painful that the patient always had to hold something in standing. She might have occasional good days when pain was almost absent. Pain was much worse after rest, particularly in the early morning. Her pain would disappear mostly if she lay down and remained still. She slept well. Her menopause occurred when she was fifty-six years old. There were six children, the youngest being thirty-five years of age. Her previous history was clear except for two curettages about the time of the menopause. There had been no injury to the hips. Her general health was good, except for an occasional sore throat.

On examination the right hip could be flexed to nearly 90° from full extension. There was no internal or external rotation and both abduction and adduction were extremely limited. From full extension she was able to flex the left hip to nearly 90°, and her rotation, abduction and adduction were practically the same as on the opposite side. In both knees there was marked grating, and other joints showed evidence of hypertrophic osteoarthritis to a degree not accounted for by her age. Her few remaining teeth were apparently healthy and her tonsils had no obvious infection. She walked with a waddling gait.

X ray examination revealed an intrapelvic protrusion of the acetabulum, such as originally described by Otto and known as an Otto pelvis. It has been more recently described as arthrokatadysis of the hip joint. There was no reaction to the Wassermann or the Neisser-Weschberg tests.

Discussion.

This condition, described originally by Otto in 1824 as an "abnormal gouty manifestation", is somewhat rare. The description as given by Otto is as follows:

> This pelvis from an adult woman is, on the whole, fairly normal in size and build. Its chief characteristic is the very deep insertion of both femoral heads in the acetabulum; so that the floors of the latter protrude far into the pelvis and are at the same time imperfectly ankylosed. The right acetabulum protrudes into the pelvis like half an orange. It forms a round, smooth, fairly thick-walled, bony shell (all diameters of the base are two and three-quarters inches broad; from that point it protrudes into the pelvis one and one-half inches). At the crest—which approximately corresponds to the centre of the acetabular fossa—is an irregular circular defect, measuring one and one-half inches probably due to absorption, initially possibly closed by membrane, but now supplying communication between the acetabulum and abdominal cavity. The lower marginal portion of the ilium protrudes like a ball into the iliac notch, due to the extended acetabulum, so that its ordinary width of about two inches, as well as the distance between the tip of the *os sacrum* and the opposite iliac margin is reduced to one inch. The acetabulum portrudes so far into the pelvis that it contains not only the head

but also the normally long neck of the femur, and the upper acetabular margin articulates with the great trochanter. The entire inner aspect of the acetabulum is smooth, also the head of the femur. Both are devoid of cartilaginous covering and have the abraded and polished appearance of some gouty joints; only the portion of the femoral head opposite the acetabular defect is roughened and eroded by absorption. The acetabular margin is rough and uneven, slightly thickened, and so constricts the femoral head that the larger head cannot be removed from the cavity of the joint—the transverse ligament of the acetabulum is ossified. (Since the roughened large trochanter grates on the roughened acetabular margin, and since the femoral neck is also somewhat angular and the small trochanter hits the sciatic tuberosity on backward flexion of the femur, the femur itself can be rotated only slightly forward and inward, but not outward and backward; thus the joint is so restricted that the abdomen and thighs form a right angle in the standing position.)

The left hip joint behaves much like the right; its acetabulum does not protrude, however, quite so far into the pelvis, but only three-quarters of an inch; the neck of the femur also has not entirely dropped down into the acetabulum, yet, since the margin is thicker than in the right hip and more constricted, and since the neck of the femur shows several protuberances at the points of friction with the acetabular margin, movement is still more restricted; the arc described by excursion of the left knee anteroposteriorly is only seven inches long; the inner bilateral femoral tuberosities can be separated only for a distance of about four or five inches. In the left hip joint the transverse ligament is ossified and is three-quarters of an inch in breadth. The right sacro-iliac symphysis is ankylosed for about one inch, and because the right femoral head is deeply inserted, the lower end of the right femur appears to be shorter than the left by one inch.

From the above described deformity and the whole appearance of the bone of this specimen, which is of interest to obstetricians, I believe the condition due to abnormal gout.

Some eighty cases are reported in the literature, and the lesion is about twice as common in females as in males, and the age incidence is about mid-life.

Many ætiological factors have been attributed to this lesion, and numerous bacteria have been regarded as causative agents, including the tubercle bacillus, the gonococcus, the streptococcus *et cetera*. Such conditions as tabes, metastatic malignant disease, *arthritis deformans*, gout, chondro-dystrophy (in which the three cartilaginous plates of the acetabulum remain unfused and are projected into the pelvic cavity) and trauma have been postulated as being actually concerned in the ætiology. It is undoubted that opinions might be easily influenced by the particular stage of the process at the time of observation, and undoubtedly, too, minor degrees and transitional forms must occur. An extensive survey of the literature on this special subject has been made by Pomeranz (*The Journal of Bone and Joint Surgery*, July, 1932) and in the following remarks the information given in his paper is fully drawn upon. An overwhelming majority of writers now believe that this condition does not constitute a disease entity, but rather is a late result of an inflammatory process, such as chronic deforming arthritis. A case is quoted in which the patient died of hæmorrhage following delivery, and a typical Otto deformity was found at autopsy; the author (Kuliga) considered the lesion to be an osteoarthritis. In a further case Cæsarean section was performed; the head of the child had a corresponding globular defect. A number of writers suggest a tuberculous basis for the condition.

Other cases have shown an intrapelvic protrusion with a definite ætiology of syphilis. A further case has been bacteriologically confirmed by isolation of a Neisserian organism. Further cases are quoted with echinococcus. The predominating characteristics are as follow: (i) a localized destructive inflammation of the acetabulum; (ii) preservation of the head and neck of the femur in the longitudinal axis; (iii) intrapelvic protrusion of the acetabulum, due to pressure from the femoral head; (iv) a rapid healing in the newly formed acetabular floor.

In 47 of the 79 cases reported this deformity occurred in females; the greatest incidence was in middle life, though one case was reported at seventy years; it has been reported as affecting persons as young as fifteen years. The diagnosis is generally made by X ray examination, which shows the fundamental changes in the acetabulum into which projects the rounded head of the femur. The degree of protrusion may vary up to five centimetres. As the acetabulum migrates, it inclines upward, inward and forward, so that it may project above the ramus of the pubic bone, encroaching upon the obturator fossa. In extreme cases it may extend to the sacro-iliac joint. The inner wall of the acetabulum may vary from shell-like to a dense eburnated wall. The external margins of the acetabulum project outwards over the neck of the femur as irregular vegetative formations. In typical cases it is important to note the preservation of the integrity of the femoral head. This would appear to be a prerequisite, since the head must maintain its boring qualities. There may, of course, be slight deviation from the general configuration of the head, sometimes a little mushroomed and at other times actually elongated. The constricted inlet, combined with the osteophytic outgrowth from the articular edge of the head, insures a definite locking of the femoral head within the acetabulum. The futility of conservative measures would be obvious. As the head of the femur submerges within the acetabulum, the trochanters approach the lateral margins of the pelvis and incline posteriorly. The greater trochanter ultimately impinges on the margins of the ilium, in the region of the acetabular shelf, and the lesser trochanter approaches the ischium. It will thus be seen why the femur cannot be rotated outwards or backwards.

In extreme cases the corresponding facets may appear round the acetabulum. The arrest of the trochanter limits the deformity and further migration of the femur is impossible. The active stage of the disease is thus terminated and the deformity is permanent.

The confusion with regard to the ætiology of this lesion appears to be due to attempts by various authors to consider it a disease entity; but the condition may be produced by any process involving essentially the acetabulum and leaving the femoral head intact. In short, any disease resulting in a localized osteomalacia of the acetabulum can produce the deformity, providing the femoral head maintains the boring qualities. The destructive process in the acetabulum must not be so severe as to prevent bone regeneration. No other observer has confirmed the findings in the one case in which a Neisserian basis was definitely established, and there are many objections to this theory as a cause for a typical Otto pelvis. Neisserian arthritis is acute and destructive, generally resulting in ankylosis or synostosis. But the typical Otto pelvis develops slowly, and there is no severely acute progressive deformity. Neisserian joints in ankylosis are often bony in character, whereas the Otto protrusion is an interlocking of small bone blocks. Neisserian infection of the hip is relatively common, while the Otto deformity is rare.

In the case of pyogenic infections the course is acute and stormy, with constitutional reaction, and ultimately results in ankylosis with loss of the typical femoral head. The infection is possibly too virulent to permit of any regenerative effort, and the femoral head is particularly involved, thus preventing any migration of the acetabulum. The boring capacity of the head must be maintained. On the other hand, attenuated infections are slowly destructive and permit of bone regeneration.

Tuberculous types have been definitely reported; but Otto pelvis occurs at an age when tuberculous hip is very uncommon.

Charcot's joint, Paget's disease, osteomalacia and metastatic malignant disease may all result in softening of the acetabulum, while the head of the femur may be unaffected and retain its boring capacity.

Pomeranz suggests two different causes of intrapelvic protrusion: (i) an acute type, probably infectious in

origin; (ii) a chronic type, occurring in any disease which results in osteomalacia of the acetabular area. In the former, Otto pelvis would not occur unless the femoral head remained intact and the protrusion developed. When the head was affected the result would be ankylosis before the deformity appeared. The second type is a more typical Otto pelvis, and the hip lesion is part of a general condition. The typical Otto pelvis is essentially a non-specific arthritis of the hip joint.

Reviews.

PSYCHO-ANALYSIS.

IN his "New Introductory Lectures on Psycho-Analysis" Dr. Sigmund Freud continues and supplements his well known introductory lectures delivered nearly twenty years ago.[1] They have been excellently translated by W. J. H. Sprott. Amidst the distinguishing marks of a great man must be included a wide understanding, modesty in his achievements and the capacity to admit previous errors. Judged by these standards, Freud is revealed as possessing these attributes in full measure. Even his severest critics must accede to an admiration of this mellowed retrospect of a long life spent in scientific battles. The book is designed not for analytic specialists, though these will read it with interest, but for the "large group of educated persons to whom, let us hope, one can ascribe a benevolent, if cautious, interest in the special value and discoveries of this young science".

It may be prophesied that this broad-minded work will do far more to popularize psycho-analysis by free association than much of the dogmatic copy which proceeds from that school of psycho-therapy.

Whilst the first chapter on the revision of the theory of dreams does little more than expand their symbolic significance, the second, on "Dreams and the Occult", is more revolutionary. Whilst examples are given to illustrate the manner in which dreams lead to an explanation of apparently occult phenomena, there is described in detail a series of incidents explicable only on the supposition that telepathy has occurred.

The section on the anatomy of the mental personality contains an excellent analogy, unfortunately too long for repetition in a review. Briefly it shows that the ego, id and super-ego must not be regarded as lying in water-tight compartments, but with certain exceptions "mixed up together". The ego, which is the part of the unconscious close to reality, has to serve three harsh masters: the id which is the deep unconscious, "a chaos, a cauldron of seething excitement"; secondly, "the super-ego which has originated conscience"; and thirdly, the external world. As the author remarks: "These demands are always divergent and often seem quite incompatible; no wonder that the ego so frequently gives way under its task."

After discussing anxiety, the instinctual life with its mingling of eros, aggressiveness and the death instinct, Freud turns to the vexed question of the psychology of woman. He regards the discovery of castration by the little girl with resultant penis envy as the turning point in her career, and states that: "three lines of treatment diverge from it; one leads to sexual inhibition or to neurosis; the second to a modification of character in the sense of a masculinity complex; and the third to normal feminity."

The importance of masturbation is regarded not as its actual effects in the pubertal stage, but rather as a symptom of faulty infantile sexuality. On the question of sexual frigidity of women, there is an admission that it is insufficiently understood and frequently due to constitutional and anatomical factors.

The chapter on explanations and applications concerns the public misconceptions concerning psycho-analysis as a branch of science and the place of dissenting schools, such as that of individual psychology, which is humorously pilloried under the caption: "If the idea were not so deuced clever, one might be tempted just to call it stupid." The reason why it falls in the latter category will intrigue the reader who sits on the fence as much as it will annoy the disciples of Adler.

Contrary to what is often believed, Freud regards his methods as having their greatest importance in research, and as a form of therapy but one among many, "though certainly *prima inter pares*". There is still abundant scope for the suggestionists and persuasionists. The possibilities of depth psycho-psychoanalysis are self-limited through the time required in each investigation.

[1] "New Introductory Lectures on Psycho-Analysis", by S. Freud, M.D., LL.D.; Authorized Translation by W. J. H. Sprott; 1933. London: The Hogarth Press; Australia: Angus and Robertson. Demy 8vo., pp. 253. Price: 16s. net.

A TEXT BOOK OF OPHTHALMOLOGY.

IT must be most gratifying to Sir John Parsons, doyen of British ophthalmology, to witness the launching of the seventh edition of his text book on "Diseases of the Eye".[1] It is now twenty-seven years since the first edition went to print, and it is doubtful if any public or private ophthalmic library in the English-speaking world does not contain one or other edition of this now world-famous treatise.

The text of the new edition follows closely that of previous editions, but the outstanding alterations and additions are as follow: First, the second chapter on physiology has been entirely rewritten to incorporate the recent researches of Sir Stewart Duke-Elder, on the production of the aqueous by dialysation and on the chemical nature of the vitreous jel.

Secondly, a detailed account of the eye examination required to determine fitness for flying both in the Royal Air Force and civil aviation is added. This section should be carefully read by all oculists, and not only those oculists whose prerogative it is to conduct such examinations.

Thirdly, a section has been added on local anæsthesia, based on the recent article by Somerville-Large in *The British Journal of Ophthalmology*.

And fourthly, many new anatomical illustrations borrowed from Eugene Wolff's recent work, "Anatomy of the Eye and Orbit", greatly enhance the value of the seventh edition.

In a work of this size, which is principally intended for students and general practitioners, much must be omitted, but in our opinion there are four omissions which in future editions might, with advantage, be made good.

In the first place no mention is made of the principles underlying all techniques for the closure of holes in detached retinæ. Secondly, the technique of intracapsular extraction of cataract and the technique of fitting contact lenses are omitted altogether. Thirdly, little stress is laid on the remarkable success obtained by the use of foreign protein in massive doses in the treatment of adult gonococcal ophthalmia witnessed not only in America, but also in Australia. And fourthly, more emphasis might have been placed on the ætiological importance of latent gonococcal urethritis in the production of acute iritis; and of the ætiological importance of disseminated sclerosis in the production of retrobulbar neuritis. This ætiological factor of disseminated sclerosis has been recently put forward most forcibly both by Adie in Great Britain, and by workers at the Mayo Clinic in America.

The opinions of Sir John Parsons are always held most highly by ophthalmic surgeons throughout the world, and the fact that he expresses scepticism on the ætiological importance of *Bacillus granulosa* in trachoma; on the use of radium and radon in *glioma retinæ*; on the wisdom of performing Holt's iridenclesis; on the use of amyl nitrite in occlusion of the central artery of the retina; and on the cauterization of disinsertions at the *ora serrata* in detachments of the retina must be given most careful consideration.

In conclusion we must strongly advocate the purchase of this book by every general practitioner and student of ophthalmology in Australia.

[1] "Diseases of the Eye", by J. H. Parsons, D.Sc., F.R.C.S., F.R.S.; Seventh Edition; 1934. London: J. and A. Churchill. Demy 8vo., pp. 703, with 21 plates and 353 text figures. Price: 18s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 22, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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AN INTERMEDIATE EYE SERVICE.

THE last few years have seen changes in medical practice that are the result of the rapid advances in medical science on the one hand and of the altered financial resources of the people on the other. It may be taken as a fundamental principle in all medical practice that the most efficient methods of treatment and the apparatus necessary to such treatment shall be available to every member of the community, no matter what his or her financial status happens to be. Certain people have the money to pay for all that they want; others have not. For the ordinary ills of mankind the number of those who can pay is not small, but those who can pay for ailments requiring the attention of specialists are less numerous. The specialist departments of public hospitals have met the needs of those who cannot pay for specialist treatment. Among those attending public hospitals, however, are many who can pay, and some of these often wish to pay something for what they receive. Even friendly society members who, by their combined efforts, are able to provide for the treatment of most illnesses, often find themselves forced to join the ranks of out-patients in special hospital departments. The specialist departments of public hospitals are becoming more and more crowded, so that the members of the staffs are overworked and find it difficult to give proper consideration to the condition of each patient. For a long time specialists have known that some change is necessary, that provision must be made for what are known as intermediate patients; and the discerning among them have realized that unless the medical profession makes plans to meet the changed conditions of society, it may have irksome or even intolerable conditions forced upon it by governments or other controlling bodies.

One of the most highly specialized branches of medicine is that dealing with disorders of the eye, and the numbers of persons in the community who need attention for ophthalmic conditions is enormous. In Great Britain the establishment of the National Ophthalmic Treatment Board has solved many difficulties. This board enables certain patients to have an ophthalmic examination by an ophthalmic medical practitioner and arranges for the provision of standard glasses, when necessary, at standard prices. A similar type of service is being inaugurated in New South Wales—what is to be known as the Medical Eye Service of New South Wales is being established by the Ophthalmological Society of New South Wales. The Ophthalmological Society of New South Wales is comprised of ophthalmologists all of whom are members of the New South Wales Branch of the British Medical Association, and the Branch has given its full approval to the formation of the medical eye service. The objects of the Medical Eye Service of New South Wales are really stated in its title. It is intended for those people "who decline to go to public hospitals and who are unable to afford private fees". Any member of the Ophthalmological Society of New South Wales may, if he so desires, become attached to the eye service. Under this service these specialists may be consulted by those who are members of the friendly societies of New South Wales and by persons whose total family income does not exceed £260 *per annum*. Medical practitioners will be able to recommend patients to the

service. A comprehensive fee of fifteen shillings is to be charged for the following attention: (i) Examination and prescription for glasses, if required, with the administration of a mydriatic if necessary. (ii) Such advice, prescription for drugs *et cetera*, and simple operative treatment as can be given at a single consultation. (iii) A report, when necessary, for the information of the general practitioner. If further consulting treatment is required, it will be continued for the first four weeks at a rate of seven shillings and sixpence per week, and thereafter at a rate of five shillings per week. When it is necessary for patients to enter hospital, all available steps will be taken to arrange for the patient's admission to public hospitals or to private hospitals in accordance with his means. Provision has been made for the supply of glasses at greatly reduced rates. The total charge for the ophthalmic surgeon's examination, advice and treatment, including the supply of glasses, ranges from twenty-five to thirty-five shillings in most cases. The Medical Eye Service of New South Wales is to be established on September 24, 1934, at De Luxe House, 267-269, Elizabeth Street, Sydney.

The announcement of the establishment of this service is made to medical practitioners with every confidence. Doubtless ophthalmic surgeons in other states will watch the new venture with the greatest interest, and doubtless also before long we shall see similar services started in other centres. No longer will any member of the community find it difficult to obtain for eye conditions adequate and expert treatment that is within his means. Those who are not in a position to pay anything will still be able to obtain treatment at public hospitals; they will not be so crowded as they are at present, for a great body of intermediate patients will have their needs supplied elsewhere. No doubt some latitude will be allowed to medical practitioners in the recommendation of patients to the service, for there must be many with large families whose incomes do not exceed £260 *per annum* by a great deal and who would be quite unable to pay the private fees of an ophthalmic surgeon. Another point on which no information has been given is whether the patient will be allowed to choose his ophthalmic surgeon from among those attached to the service; as far as possible free choice should be allowed. We predict a useful and brilliant future for the Medical Eye Service of New South Wales. Its inauguration is a most important step forwards and the Ophthalmological Society of New South Wales has made the whole community its debtor.

Current Comment.

HICCUP.

READERS of fiction may remember a story by Somerset Maugham in which a man dies at sea from intractable hiccup, to the distress of the ship's surgeon, who was at the end of his therapeutic resources. In this instance we are allowed to infer that some malign and supernatural forces were at work, but without carrying the topic into the region of the occult, we may presume that many medical practitioners have also been faced by the difficulty of treating persistent hiccup. Some years ago when epidemic hiccup was seen, apparently a larval form of encephalitis, considerable difficulty was experienced in giving relief to the sufferers from this exhausting malady, for exhausting in the extreme it is in its severer forms. E. Clark Noble, in a review of the subject, remarks that it is mentioned by the most venerable of authorities, and quotes Hippocrates and Plato.[1] Hippocrates, in one of his aphorisms, lays down that "either vomiting or singultus, or convulsions or delirium following ileus is a bad sign", thereby bracketing this apparently trifling symptom with others of recognized importance, a conjunction with which modern physicians and surgeons will readily agree. Plato gives the advice of Eryximachus to Aristophanes which is commonly given in many households today, that is, to hold the breath or to gargle or to induce sneezing. Noble goes on to summarize the anatomy and physiology of the nervous connexions of the diaphragm and points out that the phrenic nerves make connexion with the sympathetic system in the chest and that some fibres are distributed to the pleura and pericardium. Accessory phrenic nerves are said to occur in 20% to 30% of cases. Possibly variations in the exact connexions of this nerve occur, as is reasonable to suppose on clinical grounds, for patients who have the operation of phrenic section or evulsion performed under local anæsthesia give different accounts of the sensations they experience when the nerve is exposed to traction.

Noble divides the causes of singultus into the reflex (including affections of the thoracic and abdominal viscera and of the central nervous system), the epidemic, the neurotic and unknown, and the infectious and toxic. This classification is not a very satisfactory one, as it is not strictly

[1] *The Canadian Medical Association Journal*, July, 1934.

ætiological, but it serves well enough for clinical purposes. Amongst the reflex causes he places the rather rare cases in which stimulation of the phrenic nerve occurs from the irritation of some lesion in the thorax, such as lung abscess, tumour, enlarged glands, aneurysm, or inflammation of pleura or pericardium. These are less well known than those arising from lesions in the abdomen, such as ileus, peritonitis, or many varieties of obstructive or inflammatory disease or, even more familiar a cause, irritation in the gastro-intestinal tract, such as often occurs as the result of the ingestion of highly seasoned food. The nervous stimuli, that is, those arising in the nervous system itself, he also describes, such as tumour, hæmorrhage, or inflammations of diverse kind, but he does not mention another sometimes seen and due to disturbance of the appropriate segments of the spinal cord. The epidemic type has already been referred to, and the author quotes the work of Rosenow, who could find no lesion in the phrenic or vagus nerves, but demonstrated changes in the brain and cord suggestive of encephalitis, and claimed to have reproduced the corresponding condition in animals by inoculating them with naso-pharyngeal washings from infective cases. In the neurotic and unknown group he places those that occur in the course of various functional affections of the nervous system and other types that seem incapable of accurate explanation. Perhaps in this category we may place the case described by the novelist. But one of the most interesting forms of hiccup is included in the infectious and toxic group. Noble refers to a recent paper of Mayo, who voices his belief in an infective factor in the production of post-operative hiccup and possibly of other varieties also. It is interesting that only men seem to be affected in the post-operative group, and these patients are usually over the age of forty-five and not infrequently the subjects of bladder infection following catheterization, a fact that suggests the prostate as the focus. Rosenow claims to have produced diaphragmatic spasm in animals inoculated with washings of the throat from these patients, but, of course, the results of such animal experiments must be interpreted with caution. Turning from the question of causation to the practical one of treatment, it is remarked that hiccup due to simple causes yields to simple remedies, as the multiplicity of household cures will testify. But in hiccup due to upper respiratory infection local therapy is advised; the throat and then the larynx are anæsthetized with cocaine, and warm alboline is slowly dropped into the trachea. Where the hiccup is very prolonged and, whatever its cause, does not yield to other methods, the use of "Sodium amytal" is advised. A dose of seven to ten grains is given intravenously; it is designed to produce unconsciousness, and if a less amount has this effect administration is suspended. Immunity from hiccup lasts a number of hours, when the dose may be repeated, but care is needed, as the pharyngeal reflex is likely to be in abeyance also. For cases of less urgency the usual hypnotic and sedative drugs are recommended, and also nitroglycerine. In addition, there is the usual list of carminatives and anti-spasmodics and the various physical manœuvres that are sometimes successful. One simple preparation that has the personal commendation of the author is *Carbo Medicinalis*, and for the gastro-intestinal types gastric lavage is praised. High percentages of carbon dioxide in oxygen have been highly commended in the treatment of persistent hiccup, and Noble thinks the method is worthy of trial, but not always successful. Another modern development is the use of a specific antibody, which is a globulin solution prepared from a supposedly specific organism isolated by Rosenow and Mayo; this is described as effective in several cases, but the remedy is not as yet available for general use. Lastly, there is the surgical measure of interrupting the phrenic nerve. There are occasional cases in which this extreme course is justified, but it should be remembered, as the author points out, that the permanent destruction of the nerve is a procedure that is serious and not to be carried out lightly; hence in the few instances in which this radical therapeutic measure is contemplated a simple crushing of the nerve may often serve the purpose.

THE DIAGNOSIS OF ACUTE APPENDICITIS.

MOST people learn by their mistakes and many can learn by the mistakes of others. Some mistakes are excusable, some are understandable, others can be neither excused nor understood. C. W. Cutler has studied a series of errors in surgical diagnosis made at the Roosevelt Hospital, New York, during a period of three years.[1] The figures in connexion with acute appendicitis are interesting and instructive. Acute appendicitis was diagnosed 389 times; the diagnosis was incorrect in 28 instances, or 7%. Five times in the 28, acute inflammation of the tubes or tubes and ovaries was mistaken for acute appendicitis. Five times the diagnosis was acute appendicitis when the pathologist declared the inflammation to be chronic. In four instances a normal appendix was removed and the subsequent diagnosis was enteritis. On three occasions a typhlitis simulated appendicitis. Pneumonia was mistaken for appendicitis once. The two remaining mistakes that were not occasioned by unusual conditions were due to acute cholecystitis in low-lying gall-bladders. The mistakes enumerated all come within the category of those that are understandable; some of them are, of course, quite excusable. In some instances it was well nigh impossible to make a correct diagnosis. When these circumstances arise the patient must, to use Cutler's words, be given the benefit of the doubt and operation must be performed. Those to whom Cutler's paper is available are advised to read it. All medical practitioners would do well to tabulate their cases of acute appendicitis in an attempt to discover how many mistakes they have made and the reason for them.

[1] *The American Journal of the Medical Sciences*, June, 1934.

Abstracts from Current Medical Literature.

SURGERY.

Rupture of the Urinary Bladder.

RUPTURE of the bladder is always associated with a high mortality. In 1882, in a series of 166 reported cases, there was a mortality of 92%. Leon M. Bogart synopsizes the recent literature (*The American Journal of Surgery*, March, 1934) and reports six patients treated by himself. He draws attention to the increasing frequency of this incident and attributes it to motor transportation and crushing injuries. The mortality is greatly influenced by the time elapsing between injury and operative treatment. Time-consuming diagnostic methods are not favoured. Laparotomy is always favoured if the condition is suspected. The author gives differential diagnosis at length. Glucose, 5%, in normal saline solution should be given as a pre-operative measure. It is unnecessary to suture the mucosa, but two layers of muscle stitches should be used. Post-operative distension of the bladder must be prevented. The peritoneal cavity need not be drained unless gross infection is present. Cases have been reported where extraperitoneal wounds of the bladder have been treated by an indwelling catheter alone and having a low mortality; but the usual procedure consists in immediate operation and adequate drainage. One author had a mortality of 26% when operation was performed within twenty-four hours, while in delayed cases the mortality reached 75%. MacCormick, in 1886, reported the first two cases of recovery following operation for rupture of the bladder.

Traumatic Subarachnoid Hæmorrhage.

WILBUR SPRONG (*Surgery, Gynecology and Obstetrics*, April, 1934) describes the disappearance of blood from the cerebro-spinal fluid in traumatic subarachnoid hæmorrhage. The cerebro-spinal fluid has, under normal conditions, a fairly constant composition. It contains no substances not present in blood plasma, practically no colloid material, and very few cells. Red blood cells and fibrin are wholly foreign substances, and Bagley has produced experimental evidence that blood in the subarachnoid space is at once harmful and may result in permanent meningeal and nerve tissue damage. Repeated withdrawal of spinal fluid has been widely advocated for eliminating blood from the subarachnoid space. Patients with subarachnoid hæmorrhage treated by repeated lumbar punctures show a rapid disappearance of blood from the spinal fluid, and undue credit is easily given to the lumbar drainage, the rôle of processes by which Nature clears the subarachnoid space of foreign matter being overlooked. Blood disappears within five or six days from the spinal fluid. This period is not influenced by the use of repeated lumbar punctures. Xanthochromatin, which makes its gross appearance usually on the second or third day following a subarachnoid hæmorrhage, is entirely absent or reduced to a faint trace in six days, with or without lumbar punctures. The spinal fluid obtained always represents a high dilution of blood, and only a small fraction of the total fluid can be removed at one time with safety. Therefore the amount of blood eliminated by lumbar drainage is always amazingly small. In no case studied in the present investigation were more than ten drops of whole blood removed by an entire series of punctures. The rate at which blood-bearing fluid can be withdrawn from the subarachnoid space necessarily is limited to the rate of secretion of fresh fluid. This is far too slow to rival the rapid spontaneous elimination of red cells, so that only a negligible fraction of subarachnoid blood can be removed by repeated withdrawal of spinal fluid by the time fluid clearance takes place. Lumbar puncture should be used in traumatic cases for diagnosis and reducing abnormal pressure, but is entirely futile as a means of hastening the disappearance of blood from the cerebro-spinal fluid.

Disruption of Abdominal Wounds.

J. F. BALDWIN (*The American Journal of Surgery*, July, 1934) states that during a recent meeting of surgeons reports indicated that an average of from 1% to 3% of abdominal wounds undergo disruption. The author quotes the results from the Presbyterian Hospital, where, during the last eight years, 55 disruptions have occurred, in the equivalent of approximately 1% of abdominal operations. Three factors may possibly influence disruption of wounds. Probably the most important is the period during which sutures are allowed to hold the wound margins. The rate of healing and a lessening of the disruptive force are less certainly under control. Reports are quoted to show that complete digestion of the chromicized catgut used for suturing may have occurred by the third or not until the thirteenth day. A recent writer has estimated the mortality of dehiscence at 28%. Those surgeons reporting the largest incidence of disruptions generally admit that no retention sutures are used in sewing up the abdominal wounds. One recent writer has attributed disruptions to failure to close the posterior rectus sheath. The author outlines the discussion which occurred at the November, 1933, meeting of the New York Surgical Society. He reports that many surgeons removed their stay sutures as early as the seventh day, and none appear to leave them beyond the twelfth day. The author goes on to report 16,000 abdominal operations without a single disruption, his first ovariotomy being performed in June, 1886. Attention is drawn to the necessity for reinforcement by a scultetus or many-tailed bandage. During the last thirty-four years the author has allowed the sutures to remain until the fifteenth day. The wall is closed invariably with three lines of continuous sutures always reinforced by silkworm gut stay sutures which include skin, fat, aponeurosis and part of the underlying muscle. At the end of two full weeks the silkworm gut sutures are removed. For the past thirty-five years post-operative herniæ have been almost unknown in the author's practice.

Failures of Gastric Surgery.

J. A. RYLE (*The Lancet*, April 28, 1934) states that no small difficulty is encountered in determining what constitutes a failure in considering the results of gastric surgery. An anastomotic ulcer developing within a year or two of an operation for dyspepsia due to an ulcer not causing obstruction indicates a failure, for in this case the operation was not essential. If stenosis was present, then an anastomotic ulcer must be considered less of a failure, because operation was essential. If an anastomotic ulcer occurs ten years later with good health intervening, the result may be termed a success. Vicious cycle vomiting or a "dumping" stoma associated with healing of the primary ulcer is only a partial failure. Recurrence or even death following gastrectomy for a very large adherent ulcer of the lesser curvature is certainly a failure, but in this case success is very difficult of achievement. Death or recurrence of ulcer or a gastrectomy anæmia following gastrectomy for a small lesser curvature ulcer is to be regarded as a grave failure of judgement. A short circuit which gives comfort for a period of months for a pyloric carcinoma is certainly not a failure. In brief, any operation which does not alter a patient's symptoms or which results in death, where relief may have been obtained by medical measures, must be regarded as a failure. The causes of failure may be of three kinds. They may be due to technical errors or surgical inexperience; they may be due to faulty selection or judgement; whilst a minority are due to incidental misfortunes or lack of cooperation on the part of the patient. The author is familiar with five complications: (*a*) He calls the first a dumping stoma, when the small bowel becomes overloaded from the precipitate emptying of the stomach. This leads to flatulent discomforts and distension around the navel soon after a meal. To relieve this a recumbent posture should be enjoined after meals and fluids taken at least two hours after meals. (*b*) In vicious cycle vomiting there are recurring attacks of pain with copious bilious vomiting. A technical flaw is frequently responsible.

(*c*) The third complication is peri-anastomotic hæmorrhagic gastritis (Hurst). In this condition an exhibition of iron is beneficial. (*d*) Persistence of the original ulcer. (*e*) Anastomotic ulcers may cause a painful and persistent dyspepsia or may be associated with hæmorrhage, perforation, stenosis or, worst of all, gastro-jejuno-colic fistula. Neither the very old nor the very young make such good subjects for gastric surgery as those in the middle period of life. The rushed and restless business man, the medical practitioner and the man too much addicted to alcohol and tobacco are unfavourable types for gastro-jejunostomy. The duration of ulcer symptoms is an important deciding factor when operation is considered. When ulcer dyspepsia and obstructive symptoms are present for less than five years the author would hesitate to advise operation. Recent hæmorrhage and recurrent hæmorrhages are to be regarded as contra-indications to gastro-enterostomy in the absence of obstruction or the excessive size of the ulcer. Resort must be had to surgery when perforation occurs, when stenosis is present, when anchorage of the ulcer or erosion of the pancreas occurs, when chronic duodenal ulcers occur, when duodenal ileus is present, and when chronic ulcer of large size has resisted prolonged and careful medical treatment. Even in the presence of these indications secondary ulceration may also occur after gastrectomy.

A. JAMES WALTON (*ibidem*) states that every operation unnecessarily performed must be regarded as a surgical failure. An error of diagnosis is the commonest cause of such failure. No surgeon can honestly state that he has been free from this fault. This mistake is more pardonable when the presence of carcinoma is suspected. Here operation upon suspicion alone is often justified, for the consequent allaying of suspicion may set the patient on the road to recovery. In the author's experience in a series of 2,343 operations there was a percentage of 7·2 errors of diagnosis. Most of these errors were committed in the belief that carcinoma was present. It is now generally accepted that all early ulcers should be treated medically and that all chronic ulcers should be given a trial of medical treatment before surgical intervention is sought; but the more unsuitable the patient for operation, the longer the trial; and as a general rule the shorter the history of gastric symptoms, the greater the probability of carcinoma being present. The risk of operation must be carefully assessed in each patient. Many surgeons have reported the curious experience that the mortality rate rises with their experience. This is explained by the fact that a wider range of patients is referred to them for treatment. In the author's series of 2,172 gastric operations there were 703 operations performed for duodenal ulcer with a mortality of only 2%, so that the mortality of operation is less than the risk of perforation in those patients treated medically. When ulcers were present on the lesser curvature of the stomach, wedge resection and gastro-enterostomy were performed, followed by a mortality of 4·4% among 272 patients. The author regards as cured only those patients who, following operation, can eat anything and continue their daily occupation. In regard to the cures reported five years after operation, 85% of 172 patients were still symptom-free. Among the errors of technique possible are mentioned the union of the stomach to the lower ileum or even to the colon, making too small a stoma, allowing the afferent loop to become constricted by the mesocolon, making the union on the wrong side of an hour-glass constriction. Post-operative hæmorrhage is probably less easy to avoid. Retrograde intussusception cannot be guarded against. Certain complications and failures are discussed. Lewisohn published in 1925 reports of 68 cases of gastro-enterostomy in which anastonomic ulcers developed in 34%, hence many Continental surgeons now prefer the operation of partial gastrectomy. But, on the other hand, many surgeons consider the incidence of marginal ulcer to lie in the vicinity of 3%. In the author's series marginal ulcer occurred in 3·5% of gastro-enterostomies. It is directly due to hyperacidity, and this in turn is congenital and possibly a familial fault. The mortality of partial gastrectomy exceeds that of gastro-enterostomy together with the incidence of marginal ulceration. There is a much greater frequency of marginal ulceration in males. Its occurrence is directly due to hyperacidity. Partial gastrectomy is the best cure for this, but the operation is associated with a high mortality. After a properly performed gastro-enterostomy duodenal ulcers probably do not persist or recur. Perforation most commonly occurs in a chronic ulcer and it is seldom cured with a simple suture. If suture alone is possible, this patient should be closely watched and told to report for further operation if the ulcer persists. In the author's series somewhat less than 1% of patients subsequently suffered from carcinoma. In some of these the length of time intervening showed the ulcer to be malignant at the initial treatment. In some cases after operation for ulcer in the duodenum carcinoma occurred in the stomach itself, but the incidence was not higher than in normal persons.

Coronary Disease and Acute Abdominal Conditions.

WAYLAND A. MORRISON (*The Western Journal of Surgery, Obstetrics and Gynecology*, June, 1934) states that coronary disease is found to simulate an acute abdominal catastrophe in an ever-increasing number of instances. Cohnheim's teachings that coronary occlusion was always a fatal condition has been hard to disprove to the medical profession. Coronary disease is very commonly found amongst members of the profession. During 1932 and 1933, 4% of deaths in the medical profession were due to coronary disease. The disease is five times commoner in males than in females. In many clear-cut instances the surgical diseases do not enter the differential diagnosis, for the symptoms are solely anginoid and circulatory. The author reviews two patients who came under his care. In one case a labourer of forty-seven years experienced sudden epigastric pain, he vomited repeatedly, but the pain was unrelieved. Next morning the pain was very severe and radiated to the shoulders. He was examined by three physicians independently, all of whom diagnosed cholelithiasis. Symptoms recurred for seven days, when laparotomy under ethylene anæsthesia was commenced. At autopsy the abdominal organs were healthy. The right coronary artery was occluded by a thrombus, whilst the posterior aspect of the left ventricle and the posterior portion of the intraventricular septum showed recent necrosis. There was no preceding history of heart failure and there were no abnormal cardiac findings on clinical examination. The author's second patient experienced a sudden onset of epigastric pain during the night. A diagnosis of a perforation of a duodenal ulcer was made, the condition being associated with acute tenderness and board-like rigidity. The heart was normal and the temperature subnormal. Laparotomy was performed immediately, but no abnormality was found. Two months later a pericardial friction rub was present with electrocardiographic evidence of coronary disease. After a study of 141 cases of coronary disease the author found that 13% of patients experienced only epigastric pain.

Bone Graft for Non-Union of the Carpal Scaphoid.

GORDON MURRAY, of Toronto (*The British Journal of Surgery*, July, 1934), reports nine cases of non-union of the carpal scaphoid treated by insertion of a bone peg taken from the tibia. The author claims that complete function is restored and that no deformity results. The operation is carried out through a curved longitudinal incision centring over the scaphoid. With a bit measuring five-sixteenths of an inch, a hole is drilled through both fragments, the aim being to avoid damage of the cartilaginous surface. The bone peg must be flush with the surface of the fragments. A cock-up plaster splint is applied for eight weeks and by this time the bony union is complete, and within a few months even the fracture line has disappeared. The author claims that the method gives a full range of movement in all directions without pain and with normal grip. Old fractures associated with arthritis are not included in the series. It is sometimes difficult to detect a recent fracture of the scaphoid in an X ray film, but the fracture line is very readily seen later.

Medical Practice.

THE NON-DIABETIC USE OF INSULIN.

THE Ministry of Health of Great Britain prepares from time to time memoranda that are circulated for the use of its medical staff. In May, 1934, it published a memorandum that had been prepared in its Medical Intelligence Section by Dr. E. W. Adams. Copies of the memorandum were sent to Dr. J. H. L. Cumpston, Director-General, Commonwealth Department of Health. Dr. Cumpston has kindly forwarded a copy to this journal that it may be made available to medical practitioners. In publishing the memorandum *in extenso* we wish to thank Dr. Cumpston for his courtesy.

Although for some years now insulin has been employed in many non-diabetic conditions, yet, except by a somewhat arduous study of an already copious literature scattered in many languages, it is difficult for the inquirer to gain reliable information as to the value or otherwise of insulin in non-diabetic conditions. For instance, the possibility of the use of insulin in diseases other than diabetes is not even mentioned in the most recent edition of the otherwise very up-to-date manual of Doctors Beaumont and Dodds, "Recent Advances in Medicine" (1934), and, though such a use has been known for at least ten years the average text book on medicine has as yet nothing about it. Nevertheless the value of insulin in some non-diabetic states rests upon a weight of clinical evidence which cannot be ignored and which has been attested by numerous observers, some of front rank importance. Doubtless much has been written on this matter, as indeed on all medical matters, which is of little value or which is based upon insufficient experience or mal-observation, yet when all the available material has been strained through the filter of a discriminating criticism, there remains a filtrate which is worthy of analysis. It has been thought, therefore, that an account of what has been written on the subject (limited, of course, by the available material) and an attempt at some appraisal of this literature might be of value.

Nothing is said in this memorandum on the use of insulin in diabetes, since copious information on that point is readily available in every text book, as well as in various compendia, such as the work of Beaumont and Dodds already mentioned. Moreover, very useful summaries may be found in the Annual Reports of the Chief Medical Officer of the Ministry of Health for the years 1930 (pages 198-201) and 1932 (pages 72-76).

The first point which impresses a student of the literature is that although insulin has been used in the most diverse conditions,[1] so many indeed that one begins to thinks of Bishop Berkeley's Tar Water, yet the most weighty evidence of its value in non-diabetic affections is in respect of a group of diseases in which malnutrition or some fault of metabolism is either a causal factor or is a serious complication. Thus insulin has been of real value in selected cases of diseases which would appear to have so little in common as pulmonary tuberculosis, myocardial insufficiency, acidosis (especially post-operative), diphtheria and toxic goitre, to mention only a few. In none of these conditions is insulin specific, yet in all of them there is good evidence of its value in properly chosen cases and the connecting link is the metabolic one. In these states it is by virtue of its action on metabolism that the use of insulin is justified by theory and in many instances by practice also.

It looks like a truism to say that the rationale of the use of insulin in these non-diabetic states lies in its peculiar metabolic action, but there is a danger of this truism being overlooked when a scattered literature is being consulted. The mind is apt to think of insulin as being good for certain cases of pulmonary tuberculosis, good for some cardiac affections, good for acidosis, and so on, whereas the truth is that it is good for none of these affections in the sense that digitalis is good for auricular fibrillation, quinine for malaria, salvarsan for syphilis *et cetera*. It is valuable merely because it possesses a certain action upon a certain disordered form of metabolism and, no matter what the disease, there is some ground for expecting insulin to be of use if that particular form of disordered metabolism plays a prominent part in the history or course of the disease in question.

There is less evidence of the value of insulin in states in which a metabolic or nutritional defect is not so obvious. One method of grouping the material, therefore, would be to give it under two comprehensive heads: (i) the uses of insulin in diseases with underlying nutritional or metabolic defect, (ii) uses of insulin in diseases in which metabolic defect is not obvious. This method will not, however, be followed, since the second group would be so small and unimportant that, practically speaking, we should have only one group and consequently no real classification. And in any case Group (ii) could probably only exist at all because of gaps in our knowledge. The most convenient grouping will, perhaps, be an alphabetical one, that is, by grouping in alphabetical order the non-diabetic diseases in which insulin has been used. This method is not at all scientific, but has the advantage of facilitating ready reference, which is probably the desideratum of most practical importance. It may be as well, however, to begin with a consideration of the value of insulin in the treatment of malnutrition *per se*, both because the earliest non-diabetic uses of insulin were in undernourished persons and because the discussion may help towards an understanding of the rational use of insulin in states other than the diabetic.

Insulin in Malnutrition.

Pitfield in 1923 seems to have been the first to use insulin in malnutrition, and he reported excellent results in two cases of infantile inanition. He was led to employ insulin by a consideration of the often observed improved nutrition of diabetics when taking insulin. Pitfield appears, indeed, to have been the first clinician to use insulin in any non-diabetic disorder. Two years afterwards, in 1925, Falta, of Vienna, called renewed attention to the value of insulin in malnutrition. His patients were adults, namely, three very emaciated young women who had failed to gain weight despite rest in bed and careful dieting. With the aid of insulin a ravenous appetite developed and they all rapidly gained weight.

After this insulin began to be extensively tried in malnourished persons in whom no obvious cause could be found for the condition and in whom no bodily disease could be discovered. Among the more interesting of the recent papers may be mentioned those of Metz (1931), Nahum and Himwich (1932), Blotner (two papers, 1932), Lueders (1933), Barker (1933) and Belardinelli (1933). These authors all speak very favourably of the action of insulin in nutritional disorders. Lueders, for example, has been using it for as long as six years and is convinced of its value. The procedure generally adopted has been to give a small dose at first (four or five units before the principal meals) in order to test for any hypersensitivity to insulin, and then to increase the dose to ten, fifteen or even more units three times a day if the drug is well borne. When the weight has been brought up to normal the insulin may be stopped and the results are said to be lasting. It is important to instruct the patients how to recognize the commencement of symptoms due to hypoglycæmia and what to do. They should be advised to carry a supply of sugar with them. Nahum and Himwich begin with even smaller doses, three units, but they give more often, as often as every three hours in some cases, and gradually increase up to ten units.

Belardinelli's paper is noteworthy because it deals with much the same type of case as that which interested Pitfield ten years before. He describes a form of primary infantile atrophy due to a metabolic defect, which has been variously explained and which has been variously known as Parrot's atrophy, Fichelstein's decomposition and Concetti's primary atrophy. The infants have a dry

[1] Insulin has even been used as a means to attempt suicide! Beardwood (1934), for example, reports two cases which came within his own experience. Both persons recovered on prompt treatment by glucose.

skin, flaccid musculature, sunken fontanelles and eyes, are wrinkled like an old man or a monkey and have a curiously senile-looking face. The buccal mucous membrane is dry and generally covered with thrush, while there is a constant erythema around the anus, perineum and genital organs.

It was in such a case that Belardinelli obtained remarkable and completely successful results by the administration of insulin. The insulin injections were given on alternate days and were followed by injections of 20 cubic centimetres of a 30% solution of glucose. Eighteen injections of insulin were given in all and the same number of glucose injections. Improvement was noticeable after the first injection, and by the eighteenth the weight had increased from 2·5 to 4·6 kilograms. The improvement continued after cessation of the treatment, and two months after the first injection the baby weighed 7·5 kilograms and was of the appearance of a healthy, lively child, with normal colour and skin. Belardinelli states that improvement should result by the fourth or fifth injection. If it does not, the case must be regarded as unsuitable for insulin and it is no use continuing the treatment.

One of the most striking features of insulin therapy in these cases is stated to be the development of a ravenous appetite, and this should be exploited to the full. It has also been noticed that insulin increases not only appetite but gusto. The treatment is most useful in thin patients who are otherwise normal (see, however, under "Tuberculosis"). Contraindications are hypoglycæmia, vagotonia, myxœdema, active malignant states, allergic conditions, epilepsy, osteomalacia and Addison's disease. During the ingestion of insulin violent muscular work should not be undertaken.

Rationale of the Therapy.

Harris, in 1924, showed that insulin plays an important rôle in the hunger-producing mechanism, basing his observations upon cases of hyperinsulinism in which the patients suffered from an insatiable hunger. He also found, and this has been confirmed by others, that the gastric motility was increased. Previously to that Pitfield had suggested that insulin was a nutritional hormone. Metz (*loco citato*) also thinks that its action is due to a readjusted metabolism, and Lueders (*loco citato*) explained its action by its capacity to enable the cells to take up larger amounts of water, carbohydrates, fats and proteins. Blotner's second paper is especially interesting by reason of some histological researches he undertook. Microscopic examination of the fat cells showed that before treatment with insulin their walls were thick and their volume small, whereas after the insulin administration they became large and distended with thin membranous walls. There was an increase in depth of the subcutaneous fat over the abdomen. The gain in weight resulting from the treatment cannot, therefore, be explained as merely due to water retention, but is due to an actual increase in the fat deposit.

To sum up: there would appear to be no doubt that in selected cases of malnutrition, both in infants and adults, the use of insulin is of real value and, perhaps, more uniform success has been obtained in this condition than in any other, with the exception of diabetes itself. It is not suitable for indiscriminate use, however, and some of the contraindications have been mentioned above.

The employment of insulin in other conditions, arranged in alphabetical order of name, will now be considered. But it will hardly be profitable to mention all the diseases in which insulin has ever been used, for the name of those diseases is legion. This memorandum will be confined to an account of the use of the drug in those diseases in which there is some reason to believe it has been of benefit or in which its employment raises questions of especial theoretical interest.

Acidosis.

There have been several reports on the value of insulin in conditions of acidosis, especially the post-anæsthetic or post-operative variety. As early as 1923 Thalhimer reported a trial of insulin in three cases of post-operative acidosis with very gratifying results. He was led to use the drug by a consideration of the rapid disappearance of diabetic acidosis under its use. The dose he employed was five to ten units, repeated, if necessary, two or three times, and at the same time two grammes of glucose were given subcutaneously or intravenously for each unit of insulin. Glucose has, of course, long been given for acidosis, whether of the ordinary or post-operative variety, but Thalhimer states that the condition clears up much more rapidly when insulin is given as well. Langdon Brown (1927) also strongly recommended the combination. More recently Balliti (1932) recorded a very severe case of post-operative acidosis which reacted very favourably to insulin therapy. He gave twenty units daily in two equal doses, each dose being preceded by glucose administered hypodermically. Later still, Minnitt, in a communication published in 1933, warmly recommends the treatment. He infers that when post-anæsthetic toxic symptoms and acetonuria develop, they are associated with a high blood sugar. Insulin administration, which may have to be repeated, alleviates these symptoms and reduces the blood sugar. He gives it not only after anæsthesia, but sometimes also before anæsthesia as a preventive, and even during the operation itself when symptoms of shock appear.

In view of these favourable reports it would seem that insulin combined with glucose is likely to be of real value in this troublesome and sometimes alarming sequence to operative procedures.

Addiction.

Of recent years insulin has found employment in mitigating the abstinence symptoms which arise when morphine, heroin *et cetera* are withdrawn from an addict to one or another of these drugs. Hirsch (1928) stated that he had found a low blood sugar value during the withdrawal period and that the decline became more marked during the later stages. This led him to give dextrose injections and he thought that they mitigated the symptoms arising from the deprivation of the addiction drug. Anton and Jacobi (1930), acting on this hint and with a view to the more complete utilization of the sugar, tried the effect of injections of insulin and grape sugar. The insulin was given in ten unit doses and the grape sugar was administered intravenously in fifty gramme amounts in solution. Good results were claimed in one case of eucodalism and two of morphinism. Later in the same year Sakel, after first following the technique of Anton and Jacobi, finally came to rely entirely upon insulin without grape sugar, though what theoretical considerations guided him do not appear to be evident. However, his experience with fifteen addicts was that the vomiting, diarrhœa and anorexia were cut short with absolute certainty and the unrest and motor irritability were also very appreciably influenced for the better. His plan is to withdraw the morphine or other drug abruptly and give insulin for the first six to eight days of the withdrawal period, the dose being regulated by the severity of the abstinence symptoms. In some cases he used quite large doses—up to eighty units in the twenty-four hours. Larsen (1930) in Denmark also had much success with the insulin treatment of addiction and confirms Sakel's observations that the anorexia, usually observed when abrupt withdrawal of the narcotic drug is practised, is completely avoided.

More recent trials have, however, given conflicting results. While Modern (1932) reported a case in which the result of substituting "shots" of insulin instead of the morphine was highly satisfactory, Biggam and his colleagues (1932) found the results extremely disappointing in twenty addict patients in whose cases they tried the insulin plan of withdrawal. Herbrand (1933), who was himself a chronic morphine addict, relates his own experiences. He underwent several withdrawal treatments with the aid of insulin and not only did he soon relapse after each, but he did not find that the drug helped him at all by way of mitigation of the "torturing" abstinence symptoms. During his last insulin treatment, however, he found the results were very good when sulphur baths were used as an adjunct. The explanation of this is obscure.

No other conclusion, therefore, seems possible than that the value of insulin in the withdrawal treatment of mor-

phine *et cetera* is not yet fully established. The pronounced mental element in addiction makes appraisal doubly difficult, for suggestion may play an important part in contributing to the success of a new treatment. It has in fact been found that almost every new or unusual method of treatment of addiction cases has met with considerable initial success. Unfortunately, as the novelty wears off, so does the power to cure, and one is reminded of Trousseau's dictum to his students: "Make haste, gentlemen, to use a new drug while it yet has power to cure!" It may be, of course, that insulin can be of real service in some particular type of patient, but a perusal of the literature leads to a suspicion that attempts at discrimination have not been very common. Neither does the theoretic basis upon which the insulin treatment of addiction is said to rest seem very satisfying.

Arthritis (Chronic).

Howitt and Christie (1931), testing the findings of Pemberton and others that in rheumatic conditions the glucose tolerance is lowered, confirm these findings and hold that it may be accepted as a fact that sugar is not stored and used by these patients in a normal manner, either storage or oxidation being defective. They also point out that in the early stages of rheumatoid arthritis anorexia and progressive loss of weight are very common.

For these reasons they placed for some time their rheumatoid arthritis patients upon a high calorie value diet and gave insulin. Notes are given of twenty cases. The ordinary physiotherapeutic measures were also practised. The insulin course was of six weeks' duration and the initial dose was five units once a day, afterwards increased to ten units twice a day given half an hour before meals. Larger doses than this they found to give rise to disagreeable symptoms of hypoglycæmia.

The immediate results were uniformly encouraging, a gain in weight resulting in every case, and all save one felt their joints to be freer and movement not so painful. In some instances swellings subsided. No radiological changes were, however, noted.

The authors do not suggest that a cure for rheumatoid arthritis has been found. A number of patients relapsed after cessation of treatment, though in one case improvement began again when the insulin was resumed. They do suggest, however, that the malnutrition factor in rheumatoid arthritis is very important and that treatment on metabolic lines will achieve a greater measure of success than vaccines, immunization therapy and the hunt after problematical sources of infection.

Eaton and Love (1933) have obtained similar results to those of Howitt and Christie. They give details of twenty-two cases of chronic arthritis with malnutrition treated with insulin and a high calorie diet. All these showed a gain in weight of from three to twenty pounds and were improved in appetite, colour and general well-being. Concurrently with this improvement there was a decrease of pain and stiffness in the joints. They gave larger doses than Howitt and Christie, beginning with five units to be taken three times a day. The dosage was given fifteen to thirty minutes before meals, and malted milks *et cetera* were prescribed between meals. The patients were instructed as to the symptoms of threatened hypoglycæmia and how to treat them.

It will be seen that the insulin was used not for any specific effect upon the arthritis, but as a nutritional measure. The results are so interesting that further trials seem indicated.

Cancer.

In this desperate disease almost all remedies have been given a trial, and insulin is no exception. Some observers have used it in certain cases with a view to improving the general nutrition. Of special, though at present perhaps mainly of theoretical interest, are the researches of Lambert and Driessens (1933) on the action of insulin on Jensen's sarcoma. These experiments were made on two series of rats grafted with Jensen's sarcoma. One series were treated with insulin after the graft, while the control series were left untreated. The untreated rats all died within forty days with voluminous tumours. Of the insulin-treated rats 70% to 80% made a complete recovery with disappearance of the tumour, while in the remainder the development of the tumour was always slowed up. Another interesting fact observed was that injections made at a distance from the tumour were far more efficacious than those made at the periphery of the tumour.

Another interesting recent paper, which has a more immediate practical bearing, is that of Gomes da Costa (1933). In this the author details some observations he has made on the cicatrization of neoplastic cutaneous ulceration by the local use of insulin in the form of a pomade of special constitution. He gives particulars of five cases of cancerous ulceration of the nose in which the results appear to have been extraordinary. Some of these had previously resisted X ray therapy. In four of them cicatrization was complete and firm after the daily use of the pomade for periods of from thirteen to forty-six days (three of them cicatrized in thirteen, nineteen and twenty days respectively). The other case took longer. The cicatrix had remained firm up to the time of writing in all the patients, who were still under observation.

He has also made some further important observations which, if confirmed, may have a practical bearing on treatment. He finds, for example, that cancerous skin ulcers which are resistant to the action of X rays can be made sensitive to them by the previous use of insulin. He also finds that substances other than insulin which have a hypoglycæmic action (guanidin, syntalin B, codein *et cetera*) can also cicatrize neoplastic skin ulcerations on local application. The research seems to be quite a remarkable one and deserving of confirmation or confutation. Insulin in the form of a pomade or ointment has also been used as a cicatrizing agent in non-malignant ulcers, for example, ulcers of the leg, serpiginous corneal ulcer *et cetera*. Some good results are said to have been obtained.

Cardiac Affections.

Of late some interesting trials of insulin therapy have been made in cardiac affections, especially in states of cardiac insufficiency. The rationale of this treatment lies in the influence of insulin on nutrition. It is well known that glucose augments the energy of contraction of the cardiac muscle, and insulin is given with the object of facilitating the absorption of glucose by the myocardium. It was shown by Pick in 1924 that if a fragment of papillary muscle from the heart were placed in warm oxygenated bouillon, the preparation beat much more regularly and strongly on the addition of glucose. After a time, however, the beats became feebler and slower, nor did the addition of fresh glucose improve the situation. But if traces of insulin were then added the beats became powerful again and increased in frequency. Various clinicians, acting on this hint, have since treated severe cardiac insufficiency, which remained refractory to the usual measures, by glucose and insulin and seem to have obtained some remarkable results.

Osato, in 1926, appears to have been the first to publish clinical observations on this point and, among the more recent papers may be mentioned those of Kisthinios and Gomez (1931), Rimbaud *et alii* (1931), Wiener (1932), Lourenço and Sobrinho (1932) and Smith (1933). The three patients treated by Kisthinios and Gomez suffered from a very severe type of insufficiency and presented marked alternation of the pulse. No improvement resulted from the orthodox treatment. Glucose and insulin, however, completely changed the picture. The patients were, at any rate temporarily, completely relieved and the pulse alternation disappeared. A lacto-vegetarian salt-free diet was ordered and four units of insulin with eighty grammes of glucose syrup were given daily for ten days. Rimbaud and his colleagues used insulin with both glucose and lævulose and, while they do not regard the treatment as an infallible cure of all cases of cardiac insufficiency, they are convinced that it has a toni-cardiac action and is sometimes successful where ordinary measures fail. Wiener had a striking success with two of his patients, and Lourenço and Sobrinho, while not uniformly successful, obtained on the whole brilliant results with the insulin-

sugar treatment and they intend to employ it more widely in future. They used rather larger doses both of insulin and glucose than Kisthinios and Gomez, namely, twelve to twenty units of insulin daily with 100 to 150 grammes of the sugar.

Smith's paper is very interesting. He used insulin and glucose more especially in *angina pectoris*. He points out that Lewis (1931) has drawn attention to the metabolic origins of anginal pain in contradistinction to anoxæmic factors. Smith applied the therapy in the belief that a defect of the carbohydrate combustion in the heart muscle is at the root of the anginal pain and in the hope that glucose-insulin treatment might promote normal glycogen utilization and thus abrogate the pain. His method was to give five units of insulin before breakfast and before the evening meal, each dose being followed by thirty grammes of glucose taken with the meal. The duration of the treatment was from two to seventeen weeks. In six cases he obtained good results and, making every allowance for possible fallacies, he believes these results were due to the insulin-glucose therapy. He hopes the time will arrive when a more scientific selection of suitable cases for the treatment will be possible and thus occasional disappointment avoided.

Edelmann (1934) finds that in a high percentage of cases of coronary artery sclerosis and coronary thrombosis there is a latent diabetic condition and that this constitutes an important ætiological factor. In all, the author has investigated 118 cases of coronary arteriosclerosis and coronary thrombosis and in 62 of these, or 52·6%, a temporary or lasting glycosuria or hyperglycæmia was found. He advises as treatment radium irradiation of the heart area combined with the simultaneous administration of insulin.

There would thus appear to be sound reasons, both theoretical and practical, for making further trials of insulin and glucose in certain cases of cardiac disease, especially those associated with grave cardiac insufficiency.

Diphtheria.

A use for insulin has also been recently found in those very grave types of diphtheria in which the mortality is very high and which appear to be remarkably little influenced even by enormous doses of antitoxin. These severe cases appear to be increasingly frequent, and some observers claim to have been able greatly to reduce the mortality by combining glucose-insulin treatment with large doses of antitoxin.

One of the earliest and most successful trials of this new method was made by Schwentker and Noel, who reported their results in 1930. Their procedure was suggested by a consideration of the fact that if the toxæmia is severe, there is, after an initial fall in the blood sugar value, a progressive hyperglycæmia and an increasing difficulty in the assimilation of dextrose from the blood stream. This, they think, is consequent upon a suppression of the endogenous products of the pancreas. To remedy this, it occurred to them to supply glucose and make it available by insulin and so afford sufficient support to the organism to allow time for the antitoxin to neutralize the toxæmia and so avert death.

They therefore treated fourteen patients with insulin and glucose. All these were in such a toxic condition that death was imminent and several were unconscious. Thirteen of these patients recovered and in most the symptoms were very rapidly alleviated, the toxæmia disappeared and convalescence was uneventful. The technique adopted was, in brief, to administer very large doses of antitoxin, to supply fluid copiously and to give repeated injections of dextrose intravenously and ten to twenty units of insulin for each dose intramuscularly. The insulin dosage was calculated on the basis of one unit for every one to two grammes of dextrose. The more toxic the patient, the greater the need for insulin to secure complete utilization of the dextrose. The dextrose-insulin injections were repeated till the patient seemed out of danger.

Later observers, however, have not obtained such brilliant results as these, though some fairly satisfactory successes have been obtained. Benn and his colleagues (1932) have repeated Schwentker and Noel's technique with certain modifications and are convinced of its value. They lay stress upon controlling the treatment by repeated blood sugar estimations. Their cases were far more numerous than those of the earlier observers and they employed a control group to check results. Both groups were of approximately equal severity and those results were a mortality of 35·9% in the patients untreated with insulin-glucose as compared with one of 22·5% in the treated patients. (Both series, of course, had antitoxin.) The larger number investigated gives perhaps a juster conception of the possibilities of the method.

Helen Woodcock (1932) has reported her results in twenty-five very severe and toxic cases. Her experience was not nearly as favourable as that of any of the workers mentioned above, since only eight out of these twenty-five patients recovered. Still, as all were apparently in a hopeless condition, she is not inclined to condemn the treatment and, moreover, she observed that even in those patients who died there seemed to be great alleviation of the discomfort and distress. She considers that the treatment is worth while even from that point of view alone.

Another and still more recent paper is by Advier and Riou (1933), who have tried the insulin-glucose treatment in two particularly severe cases of diphtheria. These cases were stated to be of great malignity and they improved rapidly after the institution of the therapy.

Thus the results hitherto obtained have varied from brilliant to very modest success, and some of the difference may probably be accounted for by individual variations of judgement as to what constitutes a malignant case. It is as difficult to believe that all the series of cases recorded above were of comparable malignity as to believe that the differing results were due to variations of technique. "Severity" is an abstract value dependent upon the valuer—there is no objective standard. However, it would appear that on the whole glucose-insulin therapy in *diphtheria gravis* holds some promise and should be further tried out.

Gastric Diseases.

Though the evidence of the value of insulin in gastric ulcer is not altogether convincing, yet from time to time interesting reports come to hand in which the drug is stated to have been useful. One of the most informative papers is that of Feuillade (1933) and the following summary is taken from his article. It would appear that the inception of the treatment arose from the observation of Pautrier in 1924 that insulin assisted the cicatrization of ulcers of the leg. The drug was, however, first employed in stomach ulcer by Feissly in 1926. Then Glaesmer and Leoper modified the technique by giving subcutaneous injections of pepsine along with insulin. Feuillade himself relates one case in which these injections were given and he was very pleased with the result. The author does not wish to lay too much stress on one case, but he thinks the method worthy of trial in obstinate cases before thinking of surgical intervention.

Goyena (1927) also gives details of another successful case in which insulin only was used.

The sources of fallacy are, of course, numerous, and until much more material is at hand judgement must be suspended.

Another gastric disorder in which insulin is claimed to have been of benefit is the acute dilatation of the stomach sometimes met with after an operation. Mallory (1933) gives details of such a case. The acute dilatation occurred eight days after a cholecystectomy. The patient was only partially relieved by gastric lavage and the usual routine treatment, but on the administration of ten, and afterwards twenty, units of insulin daily there was rapid improvement and the patient was well after a few days. Orange juice and intravenous injections of normal saline solution with dextrose were also given.

Though not bearing directly on treatment, a paper by Labbé, Boulin and Petresco (1932) on alimentary hypoglycæmia is also worth consulting for its theoretical interest, but it does not admit of convenient summary.

Hepatic Disorders.

McCabe and Hart (1933) have treated five cases of hepatic cirrhosis by a high carbohydrate diet and as much

insulin as could comfortably be taken: five to fifteen units before each meal. Four of the patients showed much improvement and paracentesis was able to be discontinued. The authors do not offer this method as a cure for cirrhosis of the liver nor even as a permanent cure of the ascites, but they do believe that by its use compensation can be established in some cases and that in others the intervals between the tappings can be lengthened. In so hopeless a disease this would be a great gain and further reports will be awaited with interest.

In more acute diseases of the liver sugar-insulin therapy has been tried by Buresch (1932) in subacute yellow atrophy. In one case the treatment failed, but in two others the patients recovered. He started with the assumption that the liver in this disease is poor in glycogen and is consequently exposed without any protection to the attacking virus. The patients were given large quantities of lævulose and insulin, the latter in order to effect a fixation of the sugar as glycogen in the liver cells. The amounts were as much as sixty units of insulin (in three doses) and 150 grammes of dextrose daily. He concludes: "We may assume that by giving carbohydrates made assimilable by means of insulin, we not only furnish the liver with passive protection against a still unknown virus, but that by supplying this energy-producing substance in its active vital process we assist in the formation of new material." In some acute cases, however, the treatment failed and he explains this by supposing that the liver, owing to the rapid disintegration, has not the necessary time to regenerate a sufficient number of cells to insure the minimum necessary to life.

The thought arises whether it might not be worth while to institute a similar treatment in the very fatal form of liver yellow atrophy occasionally met with as a sequel to the administration of atophan and drugs of a similar class. No known treatment seems hitherto to have been of much avail.

Hyperthyreoidism and Toxic Goitre.

In the various forms of hyperthyreoidism metabolic disorders of some kind constitute an almost constant feature. This fact led Langdon Brown (1927) to advocate the use of insulin in Graves's disease and from his experience he believes the drug to be of great service in stabilizing the weight and basal metabolism. The rationale of its use he finds in the antagonistic effects of insulin and thyroxin on the blood sugar and the general metabolism. He states that it is particularly useful in cases in which the loss of weight cannot be controlled.

More recently, Kisthinios and Gomez (1931) and Cramer (1932) have reported on the use of insulin in hyperthyreoid states. The former employed it more especially to counteract the cardiac insufficiency of Graves's disease, and Cramer discusses at some length the reasons for its beneficial action in toxic goitre in general. Both seem to agree that though there is not an actual yet there is a relative insufficiency of insulin in Graves's disease. Cramer draws some interesting comparisons between toxic goitre and diabetes. In both there is a hyperactivity of the glycogenic function of the liver and there is an increased formation of carbohydrate from protein and a more rapid pouring out of sugar into the blood. But whereas in diabetes there is a rise in the blood sugar value and glycosuria, owing to impaired capacity of the organism to oxidize sugar, in hyperthyreoidism there is increased cellular activity due to a general sympathetic stimulation so that the excess sugar is suitably dealt with. By giving insulin in the latter condition, however, the cells are rested and the necessity for excessive oxidation is obviated. Thus a benefit results. Kisthinios and Gomez obtained very good results in their three cases by giving daily four units of insulin and eighty grammes of concentrated glucose syrup.

Nephritis.

The results of attempts to treat chronic nephritis with insulin have been so inconclusive that it is almost sufficient merely to mention the two papers of Mauriac *et alii* (1932) and Rathery and Rudolf (1933), which give an account of some recent trials. Mauriac and his colleagues thought that the results of some experiments they made on rabbits were sufficiently encouraging to warrant a trial on man and they gave insulin to a number of patients with chronic nephritis. The outcome was that, while still hopeful that insulin might prove to be of some use, they could not with confidence say that they obtained real improvement in any of their patients. Rathery and Rudolf indicate that the most they can say is that insulin appears to do no harm in chronic nephritis. The insulin treatment of chronic nephritis does not yet, therefore, appear even to have attained a stage of damnation by faint praise!

Obstetrics.

Vogt (1928) has written a very interesting and suggestive paper on the relation between the action of insulin and ovarian activity. His communication is largely of theoretical interest, but contains also some practical hints which might be worthy of verification.

Vogt states that he had noted that the same insulin dosage gave very varying results when administered to the same woman at different times. He satisfied himself that there was some relation between the action of insulin and the ovarian activity and that this relation was such that insulin acted most powerfully just before menstruation and least so a few days after. Thus there is a parallelism between the ovarian and insulin activities. During pregnancy the activity of insulin is subjected to no great variations. From the practical side he states that he obtained success in the treatment of ovarian hæmorrhages with insulin, for example, juvenile bleedings, climacteric bleedings and bleedings due to inflammatory states.

The difficulty in forming a just judgement on the effects of a new treatment is shown in a paper by Harding and Van Wyck (1926). These authors refer to the very successful results reported by some observers by glucose-insulin therapy in the pernicious vomiting of pregnancy. They show, however, that just as successful results are obtained by the intravenous and other use of fluids and that this is the really essential thing. In their opinion there was a misinterpretation of data, for the observers they refer to had given fluids copiously in their glucose injections and to this was due, they say, the favourable results.

Psychoses.

Though it has been stated that carbohydrate is the sole source of the energy of the brain and that when insulin is absent dextrose is converted into lactic acid as the fuel of the living brain, yet the use of insulin in the psychoses is not generally based upon such recondite theoretical considerations as these, but is merely a special application of its general action in undernourished states. As Appel, Farr and Marshall (1928) point out, critical undernutrition is a frequent problem in the treatment of psychotic patients, and the success of insulin in the therapy of certain conditions of malnutrition naturally led to its employment in psychotic patients in whom undernourishment was a prominent feature. No new principle is involved and the matter has already been sufficiently discussed under the heading of "Malnutrition". Any benefit accruing to the mental state will probably be an indirect one. The authors above-mentioned found, after trial in thirty-two mental patients who were gravely undernourished, that the insulin treatment, properly controlled, was of great value in stimulating the appetite and causing an increase of weight. Whether as a direct or indirect result of the improved nutrition, the mental state was also definitely improved in about one-fifth of the persons treated.

Sakel (1933), however, uses insulin with another object. He aims at producing a definite insulin shock by the administration of large doses. His method is first to test the insulin tolerance and then to give sufficient of the drug to produce severe hypoglycæmic shock. After this, medium doses are given for a few days. In two out of four cases of schizophrenia with a very bad prognosis there was such improvement from this procedure that the patients appeared to become normal. Sakel admits there is a certain amount of risk, but holds that this can be minimized by proper precautions and is in any case worth

taking, having regard to the severity of the disease. Other alienists, however, strongly disapprove of such heroic methods.

According to the Budapest correspondent of *The Journal of the American Medical Association* (*The Journal of the American Medical Association*, April 14, 1934, page 1244), Dr. Julius Schuster, a late assistant at the university psychoneurological clinic, asserts that insulin has both a direct and an indirect action on the central nervous system and that he has observed many mental patients were under-sensitive to insulin. He therefore advocates the use of insulin in schizophrenia and he has himself given it with highly satisfactory results to more than a hundred patients.

A more indirect use of insulin in mental disease is described by Ström-Olsen (1933). It has long been recognized that the prolonged narcosis treatment of certain mental disorders (generally with a member of the barbiturate series) is often attended by very satisfactory results, but alienists have been loath to employ it on account of its dangers. It has been found by Quastel and others that narcotics not only primarily inhibit the oxidation by the brain of substances important in carbohydrate metabolism, but also they interfere with the carbohydrate metabolism of the brain. The dangerous symptoms which not infrequently occur can, the author contends, be prevented by the administration of insulin and glucose as an integral part of the treatment.

Skin Diseases.

Some not altogether convincing reports have appeared in respect of the value of insulin in the dermatoses. Probably the remark of Azerad (1927), with more especial reference to psoriasis, still holds good: "The results are variable. The failures are more numerous than the cures and the last may perhaps be happy coincidences." A very diffuse paper by Michaelis (1929) is not without interest because, whether by coincidence or not, insulin therapy in a case of total scleroderma was followed by remarkably good results. The patient was not cured, but was very noticeably improved and the skin became much more supple. But appraisal is a little difficult because ultraviolet radiation was used as well, though Michaelis does not believe that it assisted materially in the improvement. His reasoning on this point is not very convincing. The insulin was continued for a prolonged period and scleroderma tends to improvement by time alone. Coincidence again cannot be ruled out in the case of severe urticaria treated with insulin by Wedel-Brandt (1933). Twenty-six injections of insulin in all were given and the patient had several attacks of urticaria for some time after the cessation of the treatment, which appeared at first to have failed. However, ultimately the patient remained free for four months, after which she apparently passed out of observation. The author thinks that insulin must be given the credit because the urticaria had been exceedingly troublesome for seven to eight months before the treatment and since the last attack she has not dieted or taken any special precautions. The evidence on the whole appears inconclusive.

Tuberculosis.

In selected cases of pulmonary tuberculosis in which anorexia is a prominent feature insulin seems to have been of real value. Several papers have appeared from which we select three reports by Ellman (1932), one by Heaton, also in 1932, and a paper by Allen, published December, 1933. Though insulin is held to be unsuitable in acute, active or terminal cases of pulmonary tuberculosis, yet in the chronic afebrile forms where no active disease is present, but where there is marked disturbance of nutrition, very satisfactory results have been attained by the use of carefully graduated doses of insulin with the compensatory use of glucose. It is not claimed that the treatment has any specific effect upon the disease, but the improvement is a consequence upon the better nutrition secured. Ellman urges, however, that the treatment should be controlled by clinical, radiological and bacteriological observations and that the weight, pulse, blood pressure and temperature should be studied. Fever, signs of active disease, marked hypotension and hæmoptysis are contraindications, as also are severe reactions following the injections. Both Ellman and Heaton agree that the initial doses should not exceed five units twenty minutes before the principal meals. The maximum dose advised is fifteen units twice daily twenty minutes before meals and followed three hours later by milk or glucose. Patients under treatment by insulin should also carry with them lumps of sugar for emergency use.

Allen, who has obtained some very good results with insulin in tuberculosis, does not agree that either fever or hæmorrhage is necessarily a contraindication. It may be, he says, that in far-advanced cases the insulin injections and increased feeding may raise the temperature and thus be undesirable, and it is true that the best results are usually obtained in mild and quiescent cases, but to this general rule he has encountered some striking exceptions. Hæmorrhage he would not regard as a contraindication, except in a minority of sensitive cases. His procedure also differs from that of previous observers in that he gives larger doses and for a longer period. He usually begins with five units before each meal and increases up to forty units three times a day, but "individualization should be emphasized as the chief principle". He has sometimes continued the insulin treatment for as long as eight months. The kind and quantity of the diet is also planned to suit the individual, but as a general rule a mixed diet is given containing a liberal quantity of carbohydrate, and the feeds are broken up, not only into the usual thrice daily meals, but additional meals are taken between the set ones and at bedtime.

The most striking and obvious clinical showing in a suitable case is seen in a gain in appetite, weight, strength and spirits.

In the discussion which followed Allen's paper, Nichol pointed out that there is a dearth of information as to the end results of this treatment, very few long follow-ups having been recorded. He himself followed up a number of tuberculosis patients for periods of one to five years and he found that approximately 20% of these patients maintained the weight gained, but the majority lost their gain during the first year.

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MEDICAL SERVICES IN THE NORTH AND NORTH-WEST OF AUSTRALIA.

A CHANGE is announced by the Department of Public Health of Western Australia in regard to the system under which medical services are in future to be worked in north and north-west Australia.

Hitherto medical practitioners have been appointed as district medical officers and magistrates, at places like Wyndham, Derby, Roebourne, Port Hedland, receiving a subsidy of, from £400 to £600, with the rights of private practice.

The total income is a very variable one. In some districts the results of private practice are very meagre.

Some eighteen months ago the Minister of Public Health appointed an Advisory Committee comprising representatives of the Western Australian Branch of the British Medical Association, the Pastoralists' Association, the Public Health Department and, more recently, of the Australian Inland Mission.

That Advisory Committee came to the conclusion that a medical service somewhat on the lines of that in the Northern Territory would provide better results. The idea was to secure medical men on a full-time basis who would do all medical and surgical work as well as be available for any professional duties, such as the examination of aborigines, investigations on tropical diseases and such like, that might be needed by the Commissioner of Public Health. The fees for professional services rendered to individuals would be collected by the Department. The Department will therefore take the risk of whether the net cost is greater than the present system. On the other hand, the doctor in each case knows exactly what his income will be, and he has no worry on that score or regarding the collection of patients' fees.

More recently, proposals for the constitution of certain aerial medical services have been formulated and have made considerable progress, so that it is proposed to

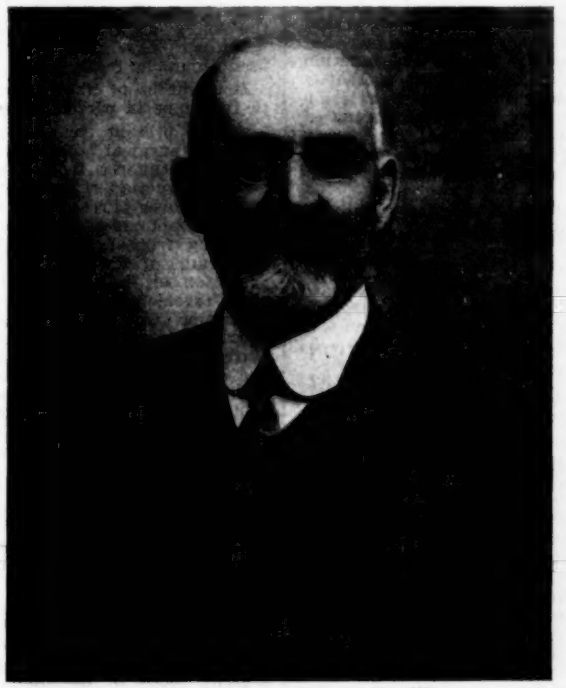
coordinate the flying services with the medical services outlined in this statement.

The salary which has been fixed for these appointments is £1,000 *per annum*, and some vacancies are already announced.

Obituary.

GEORGE THOMAS HOWARD.

DR. GEORGE THOMAS HOWARD, whose death was announced recently in these pages, was born at Long Gully, Bendigo, Victoria. He was the son of the late J. J. Howard, one of the early pioneers of Victoria. His education was begun at Sandhurst (now known as Bendigo) High School. He graduated in arts at the University of Melbourne in 1882 and took his Bachelor of Medicine and Bachelor of Surgery degrees in 1886. He became Doctor of Medicine in 1888. During his university course he did his third year arts and first and second years in medicine concurrently, a feat also accomplished by the late Sir Harry Allen. The constitution of the university was afterwards altered to prevent a repetition of this combined study, as it was thought likely to be injurious to the health of students.



In 1886 Howard was appointed to the resident staff of the Melbourne Hospital. In 1889 he became physician to Out-patients, and in 1898 was appointed in-patient physician, an appointment which he held until his retirement in 1916. In 1902 Howard was Vice-President of the Section of Medicine of the Intercolonial Medical Congress at Hobart. In 1905 he was President of the Medical Society of Victoria. In 1914 he was appointed Lecturer in Clinical Medicine at the Melbourne Hospital. After his retirement from the active staff of the Melbourne Hospital he was made consulting physician. He was one of the founders of *The Speculum*. In 1916 he became official visitor to the metropolitan hospitals for the insane. Recital of these several offices held by Howard does not give any idea of the value of his work or of the widespread influence that he wielded. He was always a keen student, and gained and held the respect of those with whom he came in contact.

During the Great War he held the rank of honorary major and did valuable work at the Base Hospital in Melbourne. In his early days he played tennis well and played cricket with medical teams against teams representing other professions. He was one of the founders and later Worshipful Master of Melbourne University Masonic Lodge. He wrote on history and many of his contributions were published in *The Argus*. He contributed some notes on empyema to the Intercolonial Medical Congress of Australasia. In the *Intercolonial Journal of Australasia* he wrote on the bath treatment of typhoid fever and on hospitals from the medical point of view. In the *Australian Medical Journal* he wrote on the diagnosis and treatment of pulmonary hydatid disease. Early in this year he wrote an interesting article that was published in this journal, on Port Phillip's early doctors. He also contributed to *The British Medical Journal* and *The Lancet*.

Dr. Felix Meyer writes:

With the passing of Dr. G. T. Howard has ended a life closely associated with the Melbourne Medical School and the Melbourne Hospital, dating back half a century. As an undergraduate at the university, taking much learning for his province, he combined the arts and medical courses, obtaining the B.A. degree while a second year medical student. Distinction that it was, it was not difficult for Howard, who had been a teacher, who had the will to study, and was gifted with a splendid memory. But he was more than academic in his outlook. He threw himself whole-heartedly into the life of the medical school, its interests, its sports. He was one of the founders and early editors of *The Speculum*; he gave it great service, it owes him much.

His professional career is dealt with elsewhere. Looking back to the long years of our friendship, he has always stood out for me as a man of unusual attainments, unassuming, without any parade of learning, of sound judgement, with a shrewd estimate of character. He had a full sense of humour and his comments always touched the subject, men or events, appositely and pleasantly, never unkindly.

Strictly ethical himself, he could not understand unethical conduct in others. He was sincere without being "dour", and though he never sought to appear impressive, he impressed by his sincerity and his broad outlook.

After his retirement from the Melbourne Hospital he devoted his leisure to his favourite study, the history of this State, and especially its early medical history. His knowledge of this subject made him the authority; he contributed interesting and informing articles to the British Medical Association and the Press, and up to the time of his last illness was collecting material for and writing up the early history of the profession in Victoria and supervising my work in this direction, a very minor part compared with his.

He enjoyed life. He loved his profession; he brought to it philosophy and sympathy. His family life was very happy. Vague and shadowy as his personality must be to the medical student of today, he will be well and affectionately remembered by those who knew him for what he was, a man of most likeable character and disposition, a fine type of physician and scholar.

TREVOR ARMSTRONG TURNER.

WE regret to announce the death of Dr. Trevor Armstrong Turner, which occurred on September 14, 1934, at Marrickville, New South Wales.

Correspondence.

ADENOHYPOPHYSEAL DYSTROPHIES.

SIR: I have read with much interest Dr. Lorimer Dods's article in THE MEDICAL JOURNAL OF AUSTRALIA of Sep-

tember 1. He has gathered together a number of very varied cases to illustrate his points. But it is impossible to accept his assumption that the hypophysis is even the chief factor in all these disturbances of growth. There is a common belief that all disturbance of growth is due to dysfunction of the hypophysis; this is surely proved fallacious by the cases of tumour of the adrenal cortex and tumours of the testes and ovaries, and probably also by lesions involving the pineal. Then there are numerous cases of delayed sexual development with bodily overgrowth up to the time of a delayed puberty and then cessation of body growth with full sexual development; there is in these cases no evidence except hypothesis that the hypophysis is involved. I should like to know if any proved case has been published where macrogenitosomia has arisen from a lesion of the hypophysis. The case described by Dr. Vickers and cited by Dr. Dods showed on autopsy a perfectly normal hypophysis and pineal, both macroscopically and microscopically. I reported a similar case in THE MEDICAL JOURNAL OF AUSTRALIA of July 25, 1931; in that case, probably a lesion in the pineal neighbourhood, there was no evidence of pituitary dysfunction, and I then pointed out that every proved case of hypophyseal origin reported in the literature, even if it showed increased body growth, exhibited depression of sexual growth and function. I am quite cognizant of the fact that some hypophyseal extracts stimulate growth and function of testes and ovaries, but the practical point is that a case of macrogenitosomia is likely to be due to a lesion not involving the hypophysis directly, but a lesion situated at some distance, usually about the pineal or quadrigeminal plate. Surely the hypophysis is but one of a chain of endocrine organs with some mutual interdependence.

Yours, etc.,

W. SPALDING LAURIE, M.D.

Melbourne,
September 8, 1934.

HYPOGLYCÆMIA AND KETOSIS: THEIR RELATIONSHIP TO CHRONIC ANTRAL DISEASE AND BRONCHIECTASIS.

SIR: Dr. Sippe's letter on the above subject in August 4 issue of the journal does not provide me with the interest anticipated. He evidently does not appreciate the reason for my criticism of his article of May 26. I object to his repeated statement, quoting his own words, "many cases of chronic antral disease, non-tuberculous pulmonary fibrosis and bronchiectasis are frequently associated with hypoglycæmia or ketosis . . ." I fail to understand how Dr. Sippe arrives at his conclusions, particularly as he only produces three case histories to support his theory and, moreover, the evidence submitted in these three instances was incomplete and incorrect, as I showed in my letter of June 16.

Dr. Sippe in his letter mentions a previous article by himself and Dr. Bostock on "Hypoglycæmic Angina". This rather surprises me, for I was under the impression that Dr. J. V. Duhig's letter criticizing their conclusions which appeared in THE MEDICAL JOURNAL OF AUSTRALIA on September 30, 1933, page 463, refuted their claims. No reply, as far as I can gather, was made to Dr. Duhig's criticism.

I protest that Dr. Sippe's claims are not supported by any scientific evidence. I can assure him that I did thoroughly study his article before writing my letter, and it was because I thought he, and not I, had rushed into print that I criticized his remarks. No mention was made in his article about a series of controls, and I assume that none was made. I feel sure that if an ordinary series of the population was investigated, the same percentage of hypoglycæmia (a low blood sugar line) would be revealed without its being considered pathological. Is Dr. Sippe's conception of hypoglycæmia based on a valid standard? Dr. Duhig showed that it was not.

Dr. Sippe misjudges when he asserts that I make light of a most difficult problem because I stated that "both sinus and chest conditions in the majority have been apparently 'cured' by the removal of septic tonsils and adenoids and adequate but conservative attention to ventilation of the diseased sinuses". For many years I have devoted serious thought to the question with which fact Dr. Sippe's Principals are well acquainted. I definitely stated in my letter that I was not thoroughly satisfied with the present line of treatment and I do not consider it the only one. The fact that I used the word "cured" in inverted commas surely shows that at least I carefully considered my statement. I did want, however, to impress upon your readers that in a large percentage of cases the treatment for the condition under discussion as suggested by me offered a very good chance of the condition being controlled. In other words, mechanics plays a big rôle in the production of the condition and its alleviation also.

As to the effect of climate upon nasal development and its influence upon the incidence of nasal accessory sinus disease and so of its sequelæ, I feel that Dr. Sippe must be familiar with my views, for I have on many occasions propounded them before his Principals.

I protest at Dr. Sippe's bringing into the discussion the term "allergy". It is inappropriate and to me it suggests the using of a term in many instances to cloak our ignorance. It is regrettable that nowadays it is being used almost as freely as the term "rheumatism".

It might be of interest to your readers in general, and to Dr. Sippe in particular, to consider the following case:

At the present time I have under my care the young son of a surgical colleague. For some time he had been a known sufferer from acidosis and symptoms of post-nasal obstruction *et cetera*. His tonsils and adenoids were removed over three years ago with no permanent relief to the symptoms of acidosis. He was again brought to me suffering from a chronic cough and a nasal discharge. Skiagrams of the sinuses showed a subacute pathological condition of the mucosa of the antra, and those of the chest revealed increased hilar and peribronchial markings. For some time he had been under glucose treatment for the acidosis. His physician suggested that this be left off. Within a very short period the cough disappeared and the nasal condition improved. X rays, although they show an improvement in the antral and chest conditions, still reveal involvement of these structures. I could claim, using the same false logic as Dr. Sippe does, that this child's condition was caused by the ingestion of glucose and the apparent cure was brought about by leaving out this article from his diet, and I could incorrectly theorize and say that "many cases of chronic antral disease, non-tuberculous pulmonary fibrosis and bronchiectasis are frequently associated with hyperglycæmia which are important pointers to a common ætiological factor, to wit, 'allergy'"!

Dr. Sippe may be interested to know that during the war period it was noticed in England that there was a great reduction in "catarrhal" illnesses among boarding school children. There was little reduction, if any, in starchy foods in the diet, but a deficiency in sugar to about half. Since the war, with the subsequent increase in sugar consumption, there has been a return to the previous state of affairs as regards these ailments. The same applies as far as teeth of children are concerned, there being more caries since than during the war period. These facts suggest that it may be the sugar that is responsible for these troubles, which is the opposite of Dr. Sippe's theory.

The theory that hypoglycæmia is the cause of many diseased states is being worked to death by a few practitioners and even recently one has suggested in an interview to the Press that it is the cause of many diseased mental states. Where will this humbug end?

In conclusion, I maintain that Dr. Sippe's evidence is not satisfactory, is very far from scientific and does not warrant his "rushing into print". He has shown lack of experience in certain aspects of his subject, and I cannot help having doubts as to the freedom from error of the other data which he puts forward in his article under discussion. Because of the illogical reasoning and poor scientific outlook displayed by Dr. Sippe, I feel it my

duty to write a letter at such length, hoping that it may prompt him to refrain in future from "rushing into print".

Yours, etc.,

R. GRAHAM BROWN.

Brisbane.
September 7, 1935.

Post Scriptum: My reply has been delayed owing to my indisposition.—R.G.B.

TRAFFIC SIGNALS.

SIR: The use of red and green signals apparently dates prior to the discovery of colour blindness by Dalton. Two worse colours could not have been selected, owing to the fact that colour blindness usually involves those colours and rarely, if ever, involves yellow and blue. About 4% to 6% of men are more or less colour blind.

But would it not be better even at this stage, at all events for motor purposes, to abandon colour signals altogether? Semaphores representing lines of white light in different positions or shapes would eliminate the colour difficulty altogether. It would be difficult to abruptly change a universal practice by land and sea, but for motor traffic the white light scheme might be easily investigated.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne,
September 11, 1934.

OVARIAN HÆMORRHAGE.

SIR: I can also supply Dr. R. Errol Maffey with the history of a further case of ovarian hæmorrhage.

On February 3, 1934, a single girl, aged twenty-one, was referred to me from a neighbouring town with a provisional diagnosis of ruptured ectopic gestation. The previous day she had complained of severe abdominal pain, mainly in the right iliac region, accompanied by a feeling of faintness.

The symptoms subsided for twenty-four hours, when she again had further abdominal pain and collapse, together with pain in the right shoulder.

When I first saw her some four hours later her pain had again subsided and her pulse rate was 80; but she was fairly tender in the right iliac fossa.

She strenuously and convincingly denied any possibility of recent pregnancy.

Opening through a mid-line incision I found a large quantity of blood in the abdominal cavity, with bleeding occurring from a ruptured cyst of the right ovary.

The ovary was excised and the patient made an uneventful recovery. The pathologist's report on the ovary reads: "With regard to the specimen of ovary (patient J.W., *ætatis* 21), the section shows hæmorrhage into the ovarian stroma and hæmorrhagic luteal cysts. There is no evidence of ectopic gestation."

Yours, etc.,

CLIVE GREER.

Ormond,
Warracknabeal,
Victoria,
September 11, 1934.

THE SWAB IN DIPHTHERIA DIAGNOSIS.

SIR: In this week's *British Medical Journal* there is a letter emanating from the Bomford (England) Isolation Hospital, which begins with the following sentence: "How much longer is the swab to be relied upon in the diagnosis of faucial diphtheria?"

A short time ago some correspondence in a similar strain appeared in THE MEDICAL JOURNAL OF AUSTRALIA, and this has prompted me to pen this letter.

After twenty-five years of experience as a pathologist in private practice I maintain that the following is sound advice in regard to the above question.

To begin with, everything depends on how the swab is taken. The swab itself must be made *secundum artem*. The wool must be tightly rolled on the stick and the distal end should be fairly pointed. To use a loosely rolled plug of wool, to merely touch the suspected spot with the broad distal end thereof, is almost useless, because, when attempting to inoculate the medium in the test tube, that spread-out distal end most likely will not come into contact with the medium at all. With a properly made swab it is the side, not the tip, which is laid on to the suspicious spot; then the stick is given a vigorous twist whereby the entire lateral surface is brought into contact with the infected spot.

Similarly, when the swab is brought into contact with the medium the side of the swab (not the tip) is laid on at the bottom end of the tube, when, by means of a twist and while dragging the swab along the surface of the medium, the entire lateral surface of the swab comes into contact with the medium.

Another fallacy lies in the fact that a certain amount of pluck and skill are required to touch the spot on the fauces, say, of a spluttering and coughing child. To turn one's head away and to shut one's eyes for fear of being bombarded with the patient's splutterings do not lead to the obtaining of a satisfactory swabbing.

Another essential lies in the conveniences offered by the laboratory undertaking the examinations. The laboratory of a clinical pathologist in private practice must be open day and night, like the surgery of the general practitioner.

If a throat swabbing arrives at 11 p.m. or any other time, somebody must be on the premises who has been trained to apply the swab correctly to the medium.

A prompt report is then the next essential. The general practitioner must receive his report before 9 a.m. the following morning, before he starts his rounds, and this must include Sundays and holidays.

Working under this scheme I have been able to give the following very definite advice to general practitioners.

Any patient clinically seriously ill with a suspicion of diphtheria must be given antitoxin at once. When not seriously ill neither patient nor a household should be alarmed before the pathologist's report has been received.

In these days of keen competition and of general depression nothing damages a general practitioner's reputation more than to become known as an alarmist, apart from putting patients to the extra expense for antitoxin injections and causing the associated mental anxiety of the household concerned, especially if the patient happens to make a complete recovery within a day or two of the first symptoms.

Yours, etc.,

ALFRED E. FINCKH, M.B. (Sydney).

227, Macquarie Street,
Sydney,
September 12, 1934.

Congresses.

THE OXFORD OPHTHALMOLOGICAL CONGRESS.

AT a recent meeting of the Council of the Oxford Ophthalmological Congress the following executive appointments were made for 1935:

Master: C. G. Russ Wood.

Immediate Past-Master: Cyril H. Walker.

Deputy Master: Philip H. Adams.

Honorary Secretary and Treasurer: F. A. Anderson, 12, St. John's Hill, Shrewsbury.

The dates for the next congress are July 4, 5 and 6, 1935.

Books Received.

ABSCESS OF THE BRAIN: ITS PATHOLOGY, DIAGNOSIS AND TREATMENT, by E. M. Atkinson, M.B., B.S., F.R.C.S.; 1934. London: Medical Publications, Limited. Royal 8vo., pp. 299, with illustrations. Price: 21s. net.

TEXT-BOOK OF MEAT INSPECTION (ANTE-MORTEM AND POST-MORTEM), INCLUDING THE VETERINARY CONTROL OF MEAT AND MEAT PRODUCTS, by R. V. Ostertag; English Edition, edited by T. D. Young, M.R.C.V.S., D.V.S.M., F.R.S.I.; translated by C. F. Marshall, M.Sc., M.D., F.R.C.S.; 1934. London: Baillière, Tindall and Cox. Royal 8vo., pp. 754, with 10 plates and 247 illustrations. Price: 45s. net.

MEDICAL ELECTRICITY FOR MASSAGE STUDENTS, by H. Morris, M.D., D.M.R.E.; 1934. London: J. and A. Churchill. Demy 8vo., pp. 397, with 103 illustrations. Price: 15s. net.

Diary for the Month.

SEPT. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 26.—Victorian Branch, B.M.A.: Council.
SEPT. 27.—South Australian Branch, B.M.A.: Branch.
SEPT. 27.—New South Wales Branch, B.M.A.: Branch.
SEPT. 28.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of the Affiliated Local Associations of Members with the Council.

Medical Appointments.

Dr. P. B. English (B.M.A.) has been appointed Government Medical Officer at Cairns, Queensland, and a Health Officer for the purposes of *The Health Acts*, 1900 to 1931.

* * *

Dr. C. J. M. Walters (B.M.A.) has been appointed Deputy Medical Superintendent, Coast Hospital, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. J. P. O'Hara (B.M.A.) has been appointed Government Medical Officer at Jandowae, Queensland.

* * *

Dr. F. W. Fay (B.M.A.) has been appointed a Member of the Board of the Hobart Public Hospitals District, Tasmania.

* * *

Dr. T. M. Gilbert (B.M.A.) has been appointed Medical Officer of Health by the Mount Margaret Road Board, Western Australia.

* * *

Dr. C. Badham (B.M.A.) has been appointed an Inspector of Theatres and Public Halls under the *Theatres and Public Halls Act*, 1908, New South Wales.

* * *

Dr. J. P. Major (B.M.A.) has been appointed a Member of the Queen's Memorial Infectious Diseases Hospital Board, Victoria.

* * *

Dr. J. M. C. Philpott (B.M.A.) has been appointed, pursuant to the *Workers' Compensation Act*, to be a Certifying Medical Practitioner at Wedderburn, Victoria.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenencies* sought, etc., see "Advertiser", pages xviii, xix

BOONAH GENERAL AND MATERNITY HOSPITAL, BOONAH, QUEENSLAND: Resident Medical Superintendent.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

MATER MISERICORDIÆ PUBLIC AND CHILDREN'S HOSPITALS, BRISBANE, QUEENSLAND: Resident Medical Officers.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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